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TASK 2-19

ANALYSIS OF LEAD IN SOIL AND DUST DATA

Allan Marcus
Task Leader

BATTELLE
Columbus Operations Arlington Office
2101 Wilson Boulevard Suite 800
Arlington VA 22201-3008

Contract 68-D0-0126

Karen Hogan, Task Manager
Edith B. Sterrett, Project Officer

Design and Development Branch
Exposure Evaluation Division
Office of Toxic Substances
U.S. Environmental Protection Agency
Washington, DC 20460

ESTIMATING RELATIONSHIPS BETWEEN SOIL LEAD, DUST LEAD, AND CHILDHOOD BLOOD LEAD: THE FEASIBILITY OF USING EXISTING DATA

Abstract

We have examined the availability of data sets from scientific studies that can establish the relationships among primary sources of lead in residential environments, lead in household dust, and childhood lead exposure in American homes. The available studies can be classified as either (1) rural small-town lead smelter and mining sites vs. urban sites, or (2) cross-sectional vs. longitudinal studies. Data at hand include a number of cross-sectional studies at rural sites, and a longitudinal study at an urban site. Data from longitudinal intervention studies at urban sites are expected to be available within the next year, and should provide much more definitive information on environmental lead pathways from lead-based paint and soil to dust. Analyses of cross-sectional data at four Western lead mining and smelter sites -- East Helena, Montana and Kellogg, Idaho in 1983, Midvale, Utah in 1989, and Butte, Montana in 1990 -- have identified some real differences among sites in the soil-to-dust pathway as well as in the bioavailability or intake of lead from soil. These analyses have shown that the relationship between household dust lead and child blood lead is almost the same in all of these communities.

Longitudinal intervention studies offer the best possibility of detecting the relative importance of lead sources and exposure pathways. However, the data from the 1979-1983 Boston study were not adequate to define a consistent picture of dust lead pathways since dust exposure was measured by lead surface loading rather than by lead concentration, and no source modification intervention was used other than uncontrolled household refinishing or remodeling. Longitudinal intervention studies in progress from which data will soon be available to us (Three-City Soil Lead Abatement Demonstration Project; Baltimore Kennedy Institute R&M Study) are much more suitable for estimating exposure pathways.

Statistical methods for analyzing these data were compared. Either of two linear equation system modeling approaches that allow for highly skewed lead concentration distributions can be used. The coefficient estimates from these structural equation models are in general consistent with estimates of soil and dust lead effects on blood lead that were previously derived by EPA (US EPA 1986, 1989a), but also allow inference about environmental pathways. The lack of data that are adequate for causal or mechanistic inference about soil-to-dust pathways is the major obstacle in predicting the effectiveness of alternative lead abatement strategies.

ESTIMATING RELATIONSHIPS BETWEEN SOIL LEAD, DUST LEAD, AND CHILDHOOD BLOOD LEAD: THE FEASIBILITY OF USING EXISTING DATA

Allan H. Marcus

1.0 INTRODUCTION AND EXECUTIVE SUMMARY

1.1 Background

Childhood lead exposure may occur from numerous environmental media, including: air, diet, drinking water, use of certain cosmetics, use of certain consumer products, and ingestion of lead-based paint and lead-contaminated soil and household dust during normal childhood hand-to-mouth contact. It is now generally agreed that the most important sources of exposure for children the U.S. in 1991 are lead-based paint and lead-contaminated soil and house dust (CDC 1991), although a large number of children still receive preventable excess lead exposure in drinking water. There are, however, substantial quantitative uncertainties about the relative importance of historic sources of lead in paint and soil. While lead-based paint was phased out after the Lead Paint Poisoning Prevention Act of 1971, an estimated 57 million housing units still contain sufficiently large quantities of lead-based paint to pose a potential hazard to current and future residents (HUD 1990). While leaded gasoline was phased out by EPA regulations for use of unleaded gasoline in the 1970's and 1980's, large quantities of lead from deposition of leaded gasoline emissions remain in soil and household dust. Industrial sources, such as non-ferrous metal smelters and battery plants, have also contributed to large local areas of soil and dust lead contamination. Any sensible program of remediation of these sources must first address the question of their relative

importance in total child lead exposure. The purpose of this report is to assess the ability of data from existing studies of childhood lead exposure in the U.S. to estimate the importance of soil, dust, and paint lead exposure.

1.2 Goals of the Study

There are four questions to be addressed:

* Are cross-sectional site-specific data adequate and available?

* Are data from longitudinal studies with interventions adequate and available?

* Are statistical methods for analysis of data from cross-sectional studies adequate to extract this information?

* Are statistical methods for analysis of data from longitudinal intervention studies adequate to extract this information?

These questions are addressed differently. The first two questions, on data availability, were answered after detailed discussions with principal investigators of the various studies. Background information on the studies can be obtained in the final report of a companion task 1-07, "Literature Search on Relationships Between Lead in Paint, Dust, and Soil." The adequacy of these data to answer the questions were assessed for each study.

1.3 Implications for Lead Abatement

Adequacy of the available data also has some important methodological implications in study design and statistical analysis. In order to estimate the efficacy of remediation or abatement programs, it is necessary to draw causal inferences about environmental lead pathways from sources to children. That is, we should like to feel some degree of confidence in statements such as:

"If we reduce lead in lead-based paint outside the house by X μg/sq.cm, then we expect a reduction of S μg/g in lead in soil 0.5 meters from the house, and a reduction in lead in dust on the floors of the house by Y μg/g or Z μg/sq.m within six months. We would then expect the blood lead level of a two-year-old child living in the house to be lower by B μg/dl after six months, and by C μg/dl after one year, due to reduction of exposure to the combination of paint, soil and dust lead. A newly born child moving into the house a year after the abatement is expected to never have a blood lead greater than A μg/dl."

This statement -- and we are paraphrasing and formalizing the goals of many proposed lead abatement projects -- has a number of implicit assumptions:

> Exterior paint lead is a significant source of dripline soil lead;

> Elimination of exterior lead paint will quickly propagate to a reduction in lead in soil at the dripline, without need for further abatement;

> Reduction of lead in soil near the house will reduce the amount of lead in household dust within six months to a year;

> Reduction of lead exposure in lead-based exterior paint, in soil near the house, and in household dust will reduce child blood lead within a few months.

The adequacy of existing data and methods for addressing these assumptions or similar assumptions in lead abatement modelling were addressed in a series of closely related studies.

1.5 Summary of Results

1.5.1 Are Cross-sectional Site-specific Data Adequate and Available?

A large number of cross-sectional data sets with household-specific soil, dust, paint, and water leads, and child blood leads, have been collected at small communities which have been the site of lead and other non-ferrous metal smelters and mines. These data were collected by CDC/ATSDR and by investigators from the Univ. of Cincinnati using generally similar sampling protocols. Most of the recent studies have been or are being conducted in connection with proposed remediation of CERCLA/RCRA (Superfund) sites and are intended to provide a "snapshot" of current conditions. Almost all of these data sets are available for detailed secondary analyses. Several statistical approaches supported by mainframe and micro-computer statistical packages are available for inferring the magnitude of hypothesized causal relationships or pathways between environmental lead sources and blood lead.

An annotated description of the data sets in hand or potentially available is given in Section 2. A description of the statistical methods that can be used to analyze these data is given in Section 3. Section 3 summarizes two detailed technical studies in Appendices A and B. Appendix A describes our methodological comparisons of structural equation models in different statistical packages, using data from three studies. Appendix B provides a brief report on our analyses for newly received data from the Butte, Montana NPL site.

Different statistical approaches were tested on four similar data sets: (1) East Helena, Montana; (2) Kellogg, Idaho; (3) Midvale, Utah; (4) Butte Montana. The results for each site were very similar by different approaches, suggesting that

available statistical methodology is adequate for inferring relationships among existing lead sources and pathways. However, some of these pathways differed substantially from one site to another. The pathway from dust lead to blood lead was almost identical in four communities. The pathway from soil lead to dust lead was strong in all four communities, but somewhat stronger in Midvale and East Helena than in Butte and Kellogg. The direct pathway from soil lead to blood lead was strong in East Helena and Midvale, weak in Kellogg, and negligible in Butte. The pathway from exterior lead-based paint to soil lead was also strong in all communities, but a direct pathway from paint to house dust was not statistically significant. However, some children with exposure to lead-based paint had elevated blood leads that may have been related to their direct ingestion of paint chips in addition to the lead paint contribution to dust and soil. These results suggest that there may be some significant child-specific, house-specific, and site-specific differences in lead exposure, sources, and pathways. Any generalization from these studies to a general or nation-wide conclusion should be done cautiously.

There are no similar environmental lead and blood lead studies for urban areas that were done after EPA's phasedown of leaded gasoline in the 1970's and early 1980's significantly reduced lead exposure in total population. Studies such as the Boston Children's Hospital study in 1980-1982, the Cincinnati Lead Program project in 1983-1991, and the Soil Lead Abatement Demonstration Projects in Baltimore, Boston, and Cincinnati in 1987-1992 were prospective longitudinal studies that were never designed as a representative cross-sectional sample and should not be compared with the Superfund site studies. These urban studies are described in Section 4 and the results of our analysis of the Boston Children's Hospital data are summarized in Section 1.5.2.

1.5.2 Are Data from Longitudinal Studies with Interventions Adequate and Available?

Longitudinal studies are particularly useful in examining the effects of changes in exposure, since each subject serves as his or her own control. In the Urban Soil Lead Abatement Demonstration Projects, there was soil lead and dust lead abatement, but no lead paint abatement. The Cincinnati dwelling units were gut rehab houses from which the lead-based paint had previously been removed. The Baltimore houses often had lead-based paint, but the exterior painted surfaces were stabilized so that recontamination of soil from exterior lead-based paint was not a factor in recontamination of the yard and house. In Boston, no lead paint determinations were made until the end of the study. The Cincinnati study had several abatement campaigns so that it will be possible to compare the effectiveness of dust abatement alone with that of combined soil and dust abatement. Long-term follow-ups in Cincinnati will also allow study of the rate of recontamination after abatement. These data are expected to be available to OSWER for preliminary evaluation and data base creation by December 15, 1991, and to other EPA offices after July 1992.

The longitudinal data from the Cincinnati Lead Program Project (supported by NIEHS) were developed to provide exposure data for a prospective study of lead effects on childhood development. The environmental data have been used for cross-sectional analyses (Bornschein et al. 1985, Clark et al. 1985). Longitudinal models for blood lead in children who did not change residence have also been carried out (Succop et al. 1987), but without consideration of environmental lead pathways and kinetics. This data set would be less useful than data from the Cincinnati Soil Lead Abatement project, which was designed as a longitudinal intervention study.

The Boston Children's Hospital study was also designed to provide exposure data for a prospective study of lead effects on childhood development. The environmental data have been used for cross-sectional analyses (Rabinowitz et al. 1985a, 1988), and identified a significant effect of household refinishing in increasing subsequent blood lead levels in one- and two-year-old children (Rabinowitz et al. 1985b). We reanalyzed these data using structural equation models for longitudinal data. The method is quite feasible, producing estimates of the importance of environmental lead pathways as well as estimates of age-dependent changes in lead exposure from soil, dust, and household refinishing. The results are discussed in Section 5, based on a detailed report in Appendix C. Since traditional methods of refinishing are known to release large quantities of bio-available lead particles (Farfel and Chisolm 1990), and since even transient exposure to these particles can significantly increase the long-term body burden of lead in the child, a significant persistent increase of blood lead following abatement was detected. However, the effect of refinishing on increasing household dust lead loadings was not significant several months later, probably due to household cleaning. Data on lead paint loading by XRF and paint chip concentration were available for less than 40 percent of the houses, and many soil lead data were missing due the difficulty of taking soil cores in the winter. Therefore the ability of these data to quantify the kinetic relationships among soil, dust, paint, and blood lead are much more limited than the Urban Soil Lead project data, where incompleteness is a much smaller problem.

1.5.3 Are Statistical Methods for Analysis of Data from Cross-sectional Studies Adequate to Extract this Information?

Relationships between blood lead and environmental lead have been derived from cross-sectional studies using linear and non-linear multiple regression relationships, as described in (Angle et al. 1984; Marcus and Cohen 1988; USEPA 1986, 1989a). However, the inter-relationships among lead concentrations in air, soil, and dust were also required in order to assess the potential long-term benefits of reducing air lead concentrations at lead point sources to a proposed new National Ambient Air Quality Standard (USEPA 1989a). An integrated approach to the simultaneous estimation of blood lead and environmental lead relationships from hypothetical causal pathway models was developed using data in three lead smelter and mining communities. The coupled system of equations was fitted using both a robust linear system method (the asymptotically distribution-free generalized least squares or AGLS method in the BMDP package EQS) and a non-linear structural equation method (PROC MODEL in the SAS/ETS package). The regression coefficients and their asymptotic standard errors were similar with either method. This suggests that several of the available statistical methods are adequate for modeling the linear or near-linear relationships among lead concentrations or surface loadings in soil, dust, paint, air, and blood. The use of separate regressions of blood lead vs. soil and dust lead, and dust lead vs. soil lead, are not recommended because the relatively large measurement errors in all of the variables will seriously attenuate the estimated regression coefficients (Fuller 1987).

1.5.4 Are Statistical Methods for Analysis of Data from Longitudinal Intervention Studies Adequate to Extract this Information?

This is a relatively unexplored subject. Certain multivariate methods such as the analysis of repeated measures with time-varying covariates, have been used to model changes in childhood IQ as a function of prenatal lead exposure and other factors (Waternaux et al. 1990). We used another variation on structural equations modeling to estimate changes in childhood blood lead and dust lead as a function of soil lead and interventions such as refinishing. The results are described in Section 5 and Appendix C. Blood lead concentrations showed a strong auto-regressive character from ages 6 to 24 months, but were also sensitive to changes in environmental lead. Environmental pathways from soil to dust to blood lead also appeared to depend on age of the child, which is difficult to explain. Missing values in the data set were awkward to deal with and much more research is needed to provide reliable methods for imputation of missing values.

1.6 Can We Establish A General Relationship Between Soil Lead and Dust Lead?

The reasons for these differences in estimates of the soil lead to dust lead pathway are not obvious. Other studies have shown that there is a strong correlation between soil lead concentration and dust lead concentration. We used both dust lead loading (mg. Pb per square meter of surface) and dust lead concentration (mg Pb per g dust) in our analyses. The dust lead concentrations in the Boston study were calculated by taking the ratio of the amount of lead in the vacuum sample to the total weight of dust, but this weight could not be accurately calculated

in very small samples and the calculated concentrations show an upward bias with decreasing weight of the dust sample. In addition to being suspected of bias, the floor lead concentrations calculated from dust lead loadings were also much worse predictors of blood lead than were the dust lead loadings.

It is known that dust cleaning can significantly reduce the loading of lead on floors without changing the concentration of lead in dust (Dolcourt et al. 1981). Additional work is needed to identify the appropriate measure of lead in house dust for use in pathway models of lead exposure. None of the studies that have attempted to directly estimate the total amount of soil and dust intake by children (Binder et al. 1986, Clausing et al. 1987, van Wijnen et al. 1990, Calabrese et al. 1989, 1990, 1991ab, and Davis et al. 1990) have explicitly adjusted these estimates for the total amount of dust in the environment. The study by van Wijnen (1990) does show that the estimated soil and dust intake varies with the environment, by comparing a campground (high intake) with a day care center (low intake).

We therefore expect to find differences in the soil lead to dust lead pathway at other urban sites as well as at rural mining and smelter sites. The differences in soil-to-dust pathways are real, though not well understood. Even if improved source identification techniques allowed estimation of the separate contributions of lead-based paint and lead in soil to household dust lead, we would still expect to find large differences in lead pathways among the housing units in a community, between communities, and possibly even differences in the soil-to-dust lead pathways between the neighborhoods within a single community. The dust lead metric used in the pathway studies should always be that one which best predicts children's blood lead, even if it is more difficult to relate this dust lead metric to soil lead.

2.0 DATA SETS FROM LEAD SMELTER AND MINING COMMUNITIES AVAILABLE FOR ANALYSIS

2.1 Butte, Montana: 1990 University of Cincinnati Study

These data were provided to us by the Principal Investigator, Dr. Robert Bornschein. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses are reported in (Bornschein et al. 1991). The available data set includes the following information for 278 children in 206 families:

IDENTIFIERS:

- Building and apartment ID;
- Sampling area (Areas A through G);
- Family ID;

ENVIRONMENTAL LEAD:

- Soil lead concentration at house perimeter;
- Curbside street dust lead concentration;
- Dust lead concentration (surface soil) outside main entrance;
- Dust lead composite concentration inside house;
- Water lead concentration in morning first-draw samples;
- Paint lead loading by XRF inside and outside the house;

BIOLOGICAL LEAD:

- Blood lead concentration;

DEMOGRAPHICS & BEHAVIOR:

Child age;
Gender;
Race;
Number of siblings under age 6;
Frequency of mouthing behavior;
Resident engaged in lead-related work;
Resident engaged in lead-related hobby;
Socio-economic status of family.

2.2 East Helena, Montana: 1983 CDC/ATSDR/Montana Department of Health & Environmental Study

These data were provided to us by the Centers for Disease Control, Center for Environmental Health and Injury Control, Lead Studies Branch, and the EPA Office of Air Quality Planning and Standards, Ambient Standards Branch. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses are reported in (Lewis and Clark et al. 1986). The EPA/OAQPS data set included additional data on X-Y coordinates and interpolated air lead concentrations that have not been merged with the CDC data. The available data sets include the following information for 396 children:

IDENTIFIERS:

Sampling area (Areas 1, 2, 3);

ENVIRONMENTAL LEAD:

Soil lead concentration (composite of front and back yard);
Dust lead composite concentration inside house;
Air lead concentration at a monitor in each area;
Presence of lead in paint on the house;

BIOLOGICAL LEAD:

Blood lead concentration;

DEMOGRAPHICS & BEHAVIOR:

Child age;
Gender;
Race;
Hours of outdoor play;
Frequency of mouthing behavior (seven categories);
Consumption of home-grown vegetables;
Resident engaged in lead-related work;
Resident engaged in lead-related hobby;
Income of family (three levels).

2.3 Granite City, Illinois: 1991 Illinois Environmental Protection Agency/ATSDR/USEPA

This study is still in progress, but Dr. Pat van Leeuwen of EPA Region 5 has expressed interest in having the ATSDR blood lead data and the Illinois/USEPA environmental data sets merged and analyzed by statistical methods and by the EPA Lead Uptake/ Biokinetic Model.

2.4 Kellogg, Idaho: 1983 CDC/EPA/Idaho Department of Health Study

These data were provided to us by the Centers for Disease Control, Center for Environmental Health and Injury Control, Lead Studies Branch. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses are reported in (CDC 1986). The available data sets include the following information for 200 children:

IDENTIFIERS:

Sampling area (Areas 1, 2, 3);

ENVIRONMENTAL LEAD:

Soil lead concentration (composite of front and back yard);
Dust lead composite concentration inside house;
Air lead concentration at a monitor in each area;

BIOLOGICAL LEAD:

Blood lead concentration;

DEMOGRAPHICS & BEHAVIOR:

Child age;
Gender;
Race;
Hours of outdoor play;
Frequency of mouthing behavior (seven categories);
Consumption of home-grown vegetables;
Resident engaged in lead-related work;
Resident engaged in lead-related hobby;
Income of family (three levels).

2.5 Leadville, Colorado: 1987 Colorado Department of Health/ ATSDR/USEPA/University of Colorado Study

These data will be provided to us by the state of Colorado in the near future. This data set has been the focus of attention and critical scrutiny by several parties because of litigation on proposed EPA cleanup of lead contamination in the community. A recent court ruling that the data will not be used in the present legal proceedings has just now freed the data for other scientific analyses not related to the litigation. We are in correspondence with Dr. Lane Cook at the Colorado DH concerning transmission of the data set to Battelle. We have also been in contact with the Univ. colorado on-site investigator, Dr. Will

Chappell, who has agreed to consult on issues relating to data collection and analysis. All identifiers relating to house location and family will be removed from the data set in order to satisfy requests for confidentiality from community residents. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses are reported in (Colorado 1988). The data set will include the following information for 233 children;

IDENTIFIERS:

Child ID w/o family or address reference;

ENVIRONMENTAL LEAD:

Soil lead concentrations in front and rear yards;
Soil lead core concentrations in play area;
Soil lead surface scrapings at play area;
Dust lead concentration (surface soil) outside main entrance;
Dust lead composite concentration on floor;
Dust lead concentration on window sills;
Water lead concentration in morning first-draw samples;
Paint lead loading by XRF in bathroom, bedroom, entry, kitchen, other interior locations;
XRF on windows, siding, exterior trim;
Recent refinishing;

BIOLOGICAL LEAD:

Blood lead concentration;

DEMOGRAPHICS & BEHAVIOR:

Child age;
Presence of nonfood mouthing behavior (9 categories);
Resident engaged in lead-related work;
Resident engaged in lead-related hobby;
Socio-economic status of family.

2.6 Leadville, Colorado: 1991 University of Cincinnati Study

This study is still in progress. The Principal Investigator, Dr. Robert Bornschein, has expressed a willingness to share a data subset adequate for studying environmental lead pathways. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses will be similar to those for the Butte study reported in (Bornschein et al. 1991). Some additional environmental data will be collected. The data set is expected to include the following information for about 250 children:

IDENTIFIERS:

- Sampling area (not household location);
- Family ID;

ENVIRONMENTAL LEAD:

- Soil lead concentration at house perimeter;
- Soil lead concentration at child play area;
- Curbside street dust lead concentration;
- Dust lead concentration (surface soil) outside main entrance;
- Dust lead composite concentration inside house;
- Dust loading inside house (mg/sq.m);
- Dustfall inside house (mg/sq.m/month);
- Lead dustfall inside house (μg/sq.m/month);
- Side-by-side dust lead loadings by wet wipe and vacuum;
- Water lead concentration in morning first-draw samples;
- Paint lead loading by XRF inside and outside the house;
- Paint condition and hazard potential;

BIOLOGICAL LEAD:

- Blood lead concentration;

DEMOGRAPHICS & BEHAVIOR:

Child age;
Gender;
Race;
Number of siblings under age 6;
Frequency of mouthing behavior (several categories);
Resident engaged in lead-related work;
Resident engaged in lead-related hobby;
Socio-economic status of family.

2.7 Midvale, Utah: 1989 University of Cincinnati Study

These data were provided to us by the Principal Investigator, Dr. Robert Bornschein. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses are reported in (Bornschein et al. 1990). The available data set includes the following information for 166 children in 128 families:

IDENTIFIERS:

House location (X,Y coordinates relative to tailings pile);
Family ID;

ENVIRONMENTAL LEAD:

Soil lead concentration at house perimeter, bare area, garden area, and child's play area;
Dust lead concentration (surface soil) outside main entrance;
Dust lead composite concentration inside house;
Water lead concentration in morning first-draw samples;
Paint lead loading by XRF inside and outside the house;

BIOLOGICAL LEAD:

Blood lead concentration;

DEMOGRAPHICS & BEHAVIOR:

Child age;

Gender;

Race;

Number of siblings under age 6;

Frequency of mouthing behavior in seven categories;

Resident engaged in lead-related work;

Resident engaged in lead-related hobby;

Socio-economic status of family.

2.8 Telluride, Colorado: 1986 University of Cincinnati Study

This data set is not yet available for reanalysis. We are pursuing further discussions with the Principal Investigator, Dr. Robert Bornschein. The total sample of 258 included 45 children with complete data for residential environmental lead, so that this study is less likely to be informative about soil and dust pathways than larger studies in communities such as Leadville.

2.9 Other Studies in Progress or Proposed: Data Availability Uncertain

Joplin, Missouri and Galena, Kansas: ATSDR/ EPA Region 7. Remedial investigations for Superfund sites. No contacts with Region 7 staff or ATSDR at this time.

Clear Creek/Central City, Colorado: Sept. 1990, Colorado Health Dept. and Univ. Colorado, for a Superfund site. Blood lead samples for 105 children, soil lead, and family interview. We are pursuing discussions with Dr. Willard Chappell, the Univ. Colo. PI.

Aspen, Colorado, Superfund Site. We are pursuing discussions with the EPA Region 8 toxicologist, Chris Weis.

Palmerton, Pennsylvania: EPA Region 8, in connection with an active secondary smelter for zinc that uses lead-contaminated

waste metal. EPA Regional toxicologist Roy Smith has provided us with preliminary blood lead data for 279 children, ages 1 to 15 years. We anticipate receiving data from a proposed large-scale child exposure assessment with about 600 children, and environmental lead variables that have not yet been defined.

3.0 STATISTICAL ANALYSES OF CROSS-SECTIONAL STUDIES AT LEAD SMELTER AND MINING SITES

3.1 Overview

Data from four Western sites were analyzed using several methods, but in almost exactly parallel model specifications. The data sets included the 1983 CDC/state studies in Kellogg, Idaho, and East Helena, Montana, and the 1989 and 1990 studies by the University of Cincinnati in Midvale, Utah and Butte, Montana. The Kellogg, East Helena, and Midvale analyses are described in detail in Appendix A. The Butte studies are described in Appendix B, All four studies had the following common characteristics:

1. Large sample sizes, 166 to 396 children under age 7;
2. Very similar sampling and analysis protocols for lead in blood, dust, soil;
3. Studies done during August or September;
4. Information on mouthing behavior and demographics was collected, but in rather different forms.

These studies were therefore regarded as the best candidates for a meta-analysis among all of the studies described in Section 2. All four studies were analyzed using the methods described above:

(i) Linear equation for blood lead, log-transformed;

(ii) Systems of linear equations for blood lead, soil lead, and dust lead, analyzed by robust AGLS method;

(iii) Systems of linear equations for blood lead, soil lead, and dust lead, log-transformed.

3.2 Soil and Dust Lead Effects on Blood Lead: Results

The blood lead equations for the structural equation methods are compared in Tables 3-1 and 3-2. Similar results for the linear system only are shown in Appendices A and B. The results in Table 3-1 from the AGLS method in EQS show some striking similarities and differences among the four sites. The first observation is that the direct relationship of soil lead to blood lead varies greatly among the four data sets. The blood lead vs. soil lead regression coefficients for East Helena and Midvale are about 2 μg/dl lead in blood per 1000 μg/g lead in soil, whereas those for Kellogg and Butte are much lower, about 0.3 to 0.5 μg/dl per 1000 μg/g. Since these data sets are independent, the differences are statistically significant.

The relationship between blood lead and dust lead concentration has two components. The first is the blood lead attributable to dust lead without adjustment for mouthing behavior, and the second is the blood lead attributable to the product of dust lead and mouthing behavior. The mouthing behavior variables are different among the studies, and have been normalized to mean value = 1 for comparison. These two components vary considerably in relative importance, with the regression coefficient on dust lead ranging from 0 in Midvale to 1.7 μg/dl per 1000 μg/g in Kellogg, and the regression coefficient of on dust lead times mouthing ranging from 0.4 in Butte to 1.7 μg/dl per 1000 μg/g in Midvale. **However, the sum of the dust lead and dust lead time mouthing regression coefficients only range between 1.6 and 2.5, and are not significantly different.** Our interpretation is that the total effect of dust lead on blood lead is about the same in all four communities, and that differences in the relative importance of these depend on differences in wording of the

interview questionnaire items dealing with mouthing behaviors, or possibly on differences in climate or community cultural practices.

The combined effect of the dust lead variables may be described by the equation

$$B1 * \text{dust lead} + B2 * \text{dust lead} * \text{mouthing} =$$

$$(B1 + B2) * \text{dust lead} * (1 + C * \text{mouthing})$$

where $C = B2 / (B1 + B2)$ describes the relative informativeness of the mouthing behavior variables for the study. The sum of the coefficients B1 + B2 is the total effect of dust lead on blood lead for a child with average mouthing behavior. These are summarized in Table 3-3 for all four studies.

The income level variable is much less predictive in the 1983 studies than was the SES variable used in the 1989-1990 studies. We suspect that parental education and household hygiene practices that affect infant and toddler lead exposure and nutrition are described better by SES than by income. There is an important increase in blood lead after age 12 months, which is most striking in the Kellogg, Idaho infants.

Similar results apply to the log-transformed linear system model in Table 3-2, but with some interesting differences. The soil lead regression coefficients are somewhat larger, and the combined dust lead + dust lead times mouthing coefficients are about equal or somewhat smaller than with the AGLS method, The overall effect of methodology does not change the significance of the soil and total dust terms, however.

3.3 Soil Lead Effect on Dust Lead: Results

The other important results of these analyses are shown in Table 3-4, in which household dust lead is related to soil lead. The results summarize more complete tables in Appendices A and B. There are some very surprising differences in the soil lead to dust lead pathway among these four communities. Not surprisingly, the contribution of soil lead to dust lead is largest around the active lead smelter in East Helena, Montana, where the contribution of soil lead to dust lead concentration is 80 to 90 percent of the soil lead concentration. The contribution of soil lead to dust lead is only somewhat smaller at Midvale, about 70 percent. The contribution of soil lead to dust lead in Butte houses is much smaller, about 26 percent, and the soil to dust lead contribution in the Silver Valley of Idaho is even smaller, less than 9 percent.

Lead-based paint was not a significant direct predictor of dust lead concentrations in Midvale or Butte houses. However, exterior XRF was a very significant predictor of elevated soil lead concentrations. Thus the pathway

Exterior lead-based paint --> Soil --> Dust --> Blood lead

appears to be a significant route of exposure, but the contribution of interior lead-based paint to house dust and to blood lead is not readily detectable from XRF data. It is obvious that some of the most highly elevated child blood leads in these communities are associated with the ingestion of deteriorating lead paint, but some of the other elevated blood leads are associated with elevated dust lead concentrations when XRF levels are low.

3.4 Discussion of Results from Lead Smelter and Mining Towns

Lead in household dust is a highly consistent predictor of elevated blood lead in four very different Western communities. The dust lead to blood lead pathway can be better predicted when the child's propensity to put non-food objects into his or her mouth (including frequent hand-to-mouth contact) is also taken into account. Since the mouthing tendency tends to decrease after age 3, many of the age-dependent effects in dust lead uptake are captured by the dust times mouthing interaction term. The combined main effect and mouthing interaction with household dust is about 2 μg/dl in blood lead per 1000 μg/g = 1 mg/g lead in house dust. It appears that fine particles in household dust are almost equally bioavailable in these communities.

Soil lead uptake appears to vary greatly among these towns, ranging from high uptake rates for children in East Helena and Midvale (greater than 2 μg/dl in blood per mg/g lead in soil) to much lower rates of uptake at Kellogg and Butte (respectively 0.4 and 0 μg/dl per mg/g). Three possibilities are:

1. Soil lead at Kellogg and Butte is much less bioavailable;
2. Child access to soil (thus soil intake) is much less at Kellogg and Butte, possibly due to greater parental awareness;
3. Soil intake is related primarily to fine surface soil particles, and the soil lead concentrations in 2-cm core samples at Kellogg and Butte are found in large particles or highly consolidated materials that are not representative of surface dust.

The available data do allow us to discriminate among these possibilities. While the first two cannot be excluded, we also know that the soil lead --> dust lead pathway at Kellogg and

Butte are much weaker than at East Helena and Midvale. This tends to support the hypothesis that the soil lead to dust lead pathway is weaker because leaded soil is not as readily broken down into surface fine particles and transported into the houses in Kellogg and Butte. The movement of surface soil into the house occurs much more readily at East Helena and Midvale. The difference in the direct uptake of soil lead into blood can also be attributed to the same hypothesis, that the soils at Kellogg and Butte have many fewer fine surface particles that can cling to children's hands and clothing. We strongly recommend that future studies of blood lead, soil lead, and dust lead include a much more complete characterization of soil particle size, chemical speciation, and soil matrix than previous studies have typically done. The potential of lead in soil to serve as a reservoir for lead in household dust should also be evaluated in any site-specific risk assessments.

These studies also confirmed that even in communities where lead deposition from historical mining and smelter activities is a very significant source of lead contamination of soil, there is also a detectable contribution to soil lead from exterior lead-based paint. Direct evidence of the importance of the exterior lead paint to soil to blood pathway was provided by stable lead isotope studies (Yaffee et al. 1983). The apparent lack of a detectable pathway from interior lead-based paint to household dust requires further study. Various methods for source identification in household dust are discussed in the report for Subtask 3 of this task. These data sets are not adequate for such identification.

Table 3-1. Comparison of System of Linear Equations
Models for Four Lead Smelter and Mining Towns
Using An Asymptotically Distribution-Free Method

	CDC 1983		Univ. Cin'ti 1989-90	
Variables	**Kellogg**	**E. Helena**	**Midvale**	**Butte**
		BLOOD LEAD EQUATION		
SOIL LEAD	0.298+	1.935***	2.199**	0.000CON
	(0.153)	(0.565)	(0.711)	(0.0125)
DUST LEAD	1.738***	0.812+	0.000CON	1.112*
	(0.578)	(0.434)	(0.007)	(0.484)
DUST LEAD* MOUTHS_ALL	0.752+	0.823**	1.665***	0.892***
	(0.451)	(0.306)	(0.569)	(0.275)
AGE0	-4.971**	-0.506NS	-1.378*	-1.419***
	(1.989)	(0.897)	(0.631)	(0.253)
INCOME (CDC 1983) or SES (1989-90)	-1.577*	0.734NS	-0.0813***	-0.0249**
	(0.767)	(0.753)	(0.0246)	(0.0100)

NOTE: Statistical significance (two-tailed)

***	means $P < 0.001$
**	means $0.001 < P < 0.01$
*	means $0.01 < P < 0.05$
+	means $0.05 < P < 0.10$ (0.05 one-tailed)
NS	means $P > 0.10$
CON	means estimate constrained to be ≥ 0.

Asymptotic standard errors in parentheses.

This model uses dummy variables for age and neighborhood (surrogate for air lead).

[1]General least squares output from EQS (from, Inter-site comparisons of environmental lead uptake. Marcus AA, Bernholc A. Battelle: Statistics and Data Analysis Systems. Research Triangle Park, NC.)

Table 3-2. Comparison of System of Linear Equations
Models for Four Lead Smelter and Mining Towns
Using SAS Proc Model with Log Transform

	CDC 1983		Univ. Cin'ti 1989-90	
Variables	**Kellogg**	**E. Helena**	**Midvale**	**Butte#**
	BLOOD LEAD EQUATION			
SOIL LEAD	0.438**	2.169***	2.435***	?.??
	(0.160)	(0.589)	(0.712)	(?.???)
	1.611**	1.097*	0.000CON	?.???
DUST LEAD	(0.522)	(0.540)	(0.---)	(?.???)
	0.412[NS]	0.607[+]	1.285*	0.?????
	(0.436)	(0.314)	(0.507)	(0.???)
DUST LEAD*	-4.501**	-0.834[NS]	-1.656*	-?.???[??]
MOUTHS_ALL	(1.617)	(0.836)	(0.666)	(0.???)
	-0.997[NS]	1.397[NS]	--0.0751**	0.???[??]
AGE0	(0.813)	(0.919)	(0.0249)	(0.???)
INCOME (CDC 1983) or SES (1989-90)				

NOTE: Statistical significance (two-tailed)

***	means $P < 0.001$
**	means $0.001 < P < 0.01$
*	means $0.01 < P < 0.05$
+	means $0.05 < P < 0.10$ (0.05 one-tailed)
NS	means $P > 0.10$
CON	means estimate constrained to be > 0

Asymptotic standard errors in parentheses.

Butte analysis in preparation

This model uses dummy variables for age and neighborhood (surrogate for air lead).

[1]Ordinary least squares option from Proc Model

[2]General least squares output from EQS (from, Inter-site comparisons of environmental lead uptake. Marcus AA, Bernholc A. Battelle: Statistics and Data Analysis Systems. Research Triangle Park, NC.)

Table 3-3. Comparison of Blood Lead vs. Dust Lead + Dust Lead * Mouthing Coefficients for Four Lead Smelter and Mining Towns Using Two Statistical Methods

Community	Method	
	AGLS	SAS Proc Model
Kellogg	2.500 (0.526)	2.023 (0.485)
East Helena	1.635 (0.386)	1.704 (0.470)
Midvale	1.665 (0.569)	1.285 (0.507)
Butte	2.004 (0.420)	?.???# (?.???)

Butte study being completed.

Table 3-4. Comparison of Dust Lead vs. Soil Lead Coefficients for Four Lead Smelter and Mining Towns Using Two Statistical Methods

Community	Method	
	AGLS	SAS Proc Model
Kellogg	0.089 (0.034)	0.087 (0.038)
East Helena	0.893 (0.100)	0.813 (0.470)
Midvale	0.717 (0.091)	0.678 (0.084)
Butte	0.266 (0.130)	?.???# (?.???)

Butte study being completed.

4.0 DATA SETS FROM URBAN AREAS IN HAND OR AVAILABLE FOR ANALYSIS IN THE NEAR FUTURE

4.1 Baltimore, Maryland: 1987-1992 Maryland Department of Environment/Superfund Soil Lead Abatement Demonstration Project (in progress)

These data will be provided to us by EPA/OSWER as soon as the data editing and evaluation are complete. OSWER expects to receive the data by Dec. 15, and to release the data for outside use by the summer of 1992. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses of the baseline data are reported in (USEPA 1991). Paint lead stabilization was carried out where necessary, and soil lead was abated in two Baltimore neighborhoods. The data set will include the following pre- and post-abatement information for about 200 children:

IDENTIFIERS:

- Child ID;
- Family or address reference;
- Neighborhood ID;

ENVIRONMENTAL LEAD:

- Soil lead concentrations in front and rear yards;
- Soil lead core concentrations in play area;
- Dust lead concentration (surface soil) outside main entrance;
- Dust lead concentration on floor in several rooms;
- Dust lead concentration on window sills;
- Water lead concentration in morning first-draw samples;
- Paint lead loading by XRF in interior locations;
- XRF on windows, siding, exterior trim;
- Hand lead wipes;

BIOLOGICAL LEAD:

Blood lead concentration;

DEMOGRAPHICS & BEHAVIOR:

Child age;

Gender;

Race;

Nutritional and medical status;

Presence of nonfood mouthing behavior (5 categories);

Resident engaged in lead-related work;

Resident engaged in lead-related hobby;

Socio-economic status of family.

4.2 Baltimore, Maryland: 1991-1992 Kennedy Institute Lead Abatement Study (in progress)

This is a longitudinal study that is intended to assess the effectiveness of several levels of lower-cost intervention in reducing child exposure to household lead-based paint and dust. The primary intervention focus is on repair of deteriorating lead-painted surfaces, trim, and windows, and maintenance of intact surfaces along with more effective control of household dust. At present only six vacant units have been studied in a pilot project, but about 150 housing units are planned for the study, with three levels of intervention. The initial data should be available by the end of 1992, and long-term effectiveness will be studied as long as continuing funding allows. Variables include:

IDENTIFIERS:

Child ID;

Family or address reference;

ENVIRONMENTAL LEAD:

Soil lead core concentrations in front and rear yards;
Soil lead scrape concentrations (surface samples);
Dust lead concentration and loading on floor, by vacuum;
Dust lead concentration and loading on window sills;
Paint lead loading by XRF in interior locations;
XRF inside and outside housing unit;

BIOLOGICAL LEAD:

Blood lead concentration;

DEMOGRAPHICS & BEHAVIOR:

Child age;
Gender;
Race;
Nutritional and medical status;
Presence of nonfood mouthing behavior;
Resident engaged in lead-related work;
Resident engaged in lead-related hobby;
Socio-economic status of family.

4.3 Boston, Massachusetts: 1987-1991 Boston Health Department of Superfund Soil Lead Abatement Demonstration Project (in progress)

These data will be provided to us by EPA/OSWER as soon as the data editing and evaluation are complete. OSWER expects to receive the data by Dec. 15, and to release the data for outside use by the summer of 1992. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses of the baseline data are reported

in (USEPA 1991). Paint lead was not measured until the end of the study. Soil lead was abated in three Boston neighborhoods. The data set will include the following pre- and post-abatement information for about 150 children:

IDENTIFIERS:

- Child ID;
- Family or address reference;
- Neighborhood ID;

ENVIRONMENTAL LEAD:

- Soil lead concentrations in front and rear yards;
- Soil lead core concentrations in play area;
- Dust lead concentration (surface soil) outside main entrance;
- Dust lead concentration on floor in several rooms;
- Dust lead concentration on window sills;
- Water lead concentration in morning first-draw samples;
- Paint lead loading by XRF at end of study;
- Hand lead wipes;

BIOLOGICAL LEAD:

- Blood lead concentration;

DEMOGRAPHICS & BEHAVIOR:

- Child age;
- Gender;
- Race;
- Nutritional and medical status;
- Presence of nonfood mouthing behavior (5 categories);
- Resident engaged in lead-related work;
- Resident engaged in lead-related hobby;
- Socio-economic status of family.

4.4 Boston, Massachusetts: 1979-1983 Boston Children's Hospital/ Boston Hospital for Women Prospective Lead Study

These data were provided to us by one of the Principal Investigators, Dr. Michael Rabinowitz. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses of the environmental and blood lead data are reported in (Rabinowitz et al. 1984, 1985ab, 1988). Paint lead was measured in only a fraction of the houses, about 40 percent by XRF and 10 percent by AAS analysis of paint chips. Only 19 of 250 houses had both paint XRF and AAS paint chip data. Soil lead was measured when children were 18 and 24 months of age but many values were missing since samples could not be obtained during the winter. Dust lead loadings on floors, window sills, and furniture were measured from vacuum samples at 1, 6, 18, and 24 months of age, but the concentrations could not be accurately calculated when then amount of dust collected was too small. Blood leads were measured in umbilical cord blood and at ages 6, 12, 18, and 24 months. The data set provided to us did not have hand lead, pica, or date of birth, but we are requesting these additional data. Information on refinishing or moving within the last six months was very complete. We used the following information for 142 children who did not change residence during the study:

IDENTIFIERS:

Child ID;

ENVIRONMENTAL LEAD:

Soil lead core concentrations at 18 and 24 months;
Dust lead loading at ages 1, 6, 18, and 24 months;
Dust lead concentration at age 1, 6, 18, and 24 months;
Dust lead loadings on window sills at ages 1, 6, 18, 24 months;
Water lead concentration in partially flushed samples;
Paint lead loading by XRF or concentration by AAS;
Refinishing within last six months;
Lead in formula and breast milk at ages 1 and 6 months;

BIOLOGICAL LEAD:

Blood lead concentration at ages 6, 12, 18, and 24 months;

DEMOGRAPHICS & BEHAVIOR:

Child age;

4.5 Cincinnati, Ohio: 1987-1992 University of Cincinnati/Superfund Soil Lead Abatement Demonstration Project (in progress)

These data will be provided to us by EPA/OSWER as soon as the data editing and evaluation are complete. OSWER expects to receive the data by Dec. 15, and to release the data for outside use by the summer of 1992. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses of the baseline data are reported in (USEPA 1991). Paint lead was not a factor in this study since the selected units were mostly gut rehabs without lead-based paint. Soil lead was abated in community areas, and street dust was cleaned up in five Cincinnati neighborhoods. The data set will include the following pre- and post-abatement information for about 200 children:

IDENTIFIERS:

Child ID;

Family or address reference;

Neighborhood ID;

ENVIRONMENTAL LEAD:

Soil lead concentrations in defined zones (e.g. 25-m concentric rings around housing unit);

Soil lead core concentrations in play area;

Dust lead composite concentration in several rooms;

Street dust lead concentrations;

Water lead concentration in morning first-draw samples;

Hand lead wipes;

BIOLOGICAL LEAD:

Blood lead concentration;

DEMOGRAPHICS & BEHAVIOR:

Child age;

Gender;

Race;

Nutritional and medical status;

Presence of nonfood mouthing behavior (9 categories);

Resident engaged in lead-related work;

Resident engaged in lead-related hobby;

Socio-economic status of family.

4.6 HUD Abatement Demonstration Study: 1989-1990, in Baltimore MD, Washington DC, Seattle-Tacoma WA, Denver CO, Indianapolis IN, and Birmingham AL. Follow-Up Study (CAPS) in Progress

169 vacant single-family houses were given various levels of lead paint and house dust abatement. As these units are re-occupied, the recontamination or further decontamination of the house will be assessed.

ENVIRONMENTAL VARIABLES:

Lead paint loading, interior and exterior, by XRF;
Lead paint chip concentration by AAS;
Amount of lead-painted surface area;
Soil lead core sample;
House dust lead loading by wet wipe.

4.7 HUD National Lead Survey: 1989-1990 in 30 Counties in 48 Contiguous States

This was a survey designed to be representative of the US housing stock. A large number of measurements were made in wet rooms and dry rooms of each of 381 dwelling units, of which 284 were privately owned. Variables include:

ENVIRONMENTAL LEAD

Soil lead core concentration;
Dust lead concentration and loading in vacuum samples;
Interior and exterior lead paint by XRF.

4.8 St. Louis, Missouri: 1980-1983 Lead Paint Abatement Effectiveness Study

Data on blood leads before and after abatement were provided by the C.G. Copley, the Assistant Director of the St. Louis Health Department. Soil lead concentrations in the same census tracts were also provided to us, but have yet to be linked with the residence locations, although Mr. Copley believes that there is enough data in the records to do this. This would provide a less adequate measure of potential contributions to blood lead and pre and post-abatement blood lead from soil lead and from a component of dust lead attributable to soil lead.

IDENTIFIERS:

Child ID;

ENVIRONMENTAL LEAD:

Soil lead concentrations in defined zones (census tract);

BIOLOGICAL LEAD:

Blood lead concentration before and after abatement;

Erythrocyte protoporphyrin as an index of long-term exposure;

DEMOGRAPHICS & BEHAVIOR:

Child age;

Compliance with scheduled clinic visits (yes/no).

4.9 Other Studies in Progress or Proposed: Data Availability Uncertain

Cincinnati OH, University of Cincinnati: Principal Investigator, Scott Clark. Study of the rate of infiltration of lead into house dust from lead-based paint and soil lead. Supported by EPA/ORD/ECAO.

Jersey City NJ, Robert Wood Johnson Medical School: Principal Investigators George Rhoads and Paul Lioy. Longitudinal study of the effectiveness of household dust control and parental education in reducing childhood lead exposure from paint and soil. Children will be recruited pre-natally. Study design includes both positive and negative controls to assess the behavioral effect of interaction of family or caretaker with the project staff. Supported by EPA/ORD/AREAL.

Baltimore MD, Baltimore City Health Department: scientific support by Mark Farfel and Kennedy Institute. Longitudinal study of children in houses scheduled for lead paint abatement under Maryland law. Additional support for environmental and blood lead follow-up and monitoring, from CDC/CEHIC/Lead Studies Branch.

Boston MA, Boston Health Department: Longitudinal study of children in houses scheduled for lead paint abatement under Massachusetts law. Additional support for environmental and blood lead follow-up and monitoring, from CDC/CEHIC/Lead Studies Branch.

5.0 STATISTICAL ANALYSES OF THE BOSTON CHILDREN'S HOSPITAL LONGITUDINAL STUDY

5.1 Overview

The data from the 1979-1983 prospective lead exposure study were provided to us by Dr. Michael Rabinowitz. These blood lead and environmental lead data were intended primarily to provide exposure assessment information for use as predictors of neurobehavioral deficits in infants and young children (Bellinger et al., 1985, 1987, 1989), but have also been extensively analyzed to provide estimates of post-natal lead exposure (Rabinowitz et al. 1984, 1985ab, 1988). The time pattern of environmental lead data collection was not ideal for a time series study, and there are so many missing values in the data that some formal approach is clearly needed to establish the effects of missing data on the analyses. The method we used was to assign dummy variable codes for missing variables whose absence left a gap in the longitudinal pathway models. If all cases with missing data were eliminated for the 153 children who did not change residence, then we would have had only 61 subjects out of 153. Including a few such dummy-variable codings increased the number of useable cases to 142. The significant correlations between the missing value codes and some of the non-missing variables established that the data were not missing completely at random, and the implications of this finding need to be evaluated. Several longitudinal structural models of blood lead and dust lead provided a statistically satisfactory fit to the data. The models were based on plausible prior hypotheses about mechanisms and pathways, so that this modelling exercise is more than simple curve-fitting.

5.2 Effects of Environmental Lead on Blood Lead: Results

The results for the best-fitting model are shown in Table 5-1. Blood lead at ages 12, 18, and 24 months could be predicted by blood lead six months earlier, with a surprisingly consistent auto-regression parameter of about 0.4 (correlation time scale or mean blood lead residence time of 6.5 months). This long persistence could mean that there was already a significant mobilizable body burden of lead in the skeleton or other tissues, and that a substantial part of lead levels in blood are due to endogenous lead pools. Another possibility is that lead exposure by a set of relatively constant sources produces a high autocorrelation. We are inclined to disbelieve the hypothesis of constant exposure, since air lead and dust lead levels dropped very rapidly from 1980 to 1982 or 1983. The autocorrelation of blood lead at six-month intervals is thus more likely to be associated with responses to rapidly changing air lead and dust lead levels which were caused, in part, by EPA's leaded gasoline phasedown during this period.

It is more important to note that changes in blood lead were associated with changes in lead exposure. The effect of household refinishing on blood lead was striking, with an increase in blood lead of about 3 μg/dl at ages 12 and 18 months, and 1.6 μg/dl at 24 months. As seen in Table 5-1, the effect of dust lead loading on blood vary with age, but dust lead is associated with large and statistically significant increases in blood lead at ages 18 and 24 months. The increase of blood lead with increasing soil lead concentration is largest and most significant at ages 12 and 18 months. However, the soil lead term in the blood lead equations is a direct effect. The indirect effect of soil lead depends on the soil lead to dust lead pathway.

5.3 Soil Lead Effect on Dust Lead: Results

Two models on the autocorrelation of dust lead were tested. Model 1 assumed that dust lead loadings were also autocorrelated, with high levels at age 6 months predicting higher-than-average levels at 18 months, and high levels at 18 months predicting higher-than-average dust lead loadings at age 24 months. The 18- to 24-month autoregression was small (0.10 in Table 5-2) but statistically significant, whereas the 6- to 18-month autoregression was much smaller and not significant. The regression coefficient of 18-month dust lead loading on soil lead concentration was also highly significant, establishing that soil lead had an indirect pathway to blood lead through household dust.

In Model 2, the autocorrelation of dust leads was dropped under the hypothesis that house floors were cleaned sufficiently often in this sample of predominantly well-educated middle-class families to offset any long-term persistence of dust lead loadings on the floor. Model 2 did not fit the data as well as Model 1. In addition, the regression coefficient of dust lead loading on soil lead was not statistically significant at age 24 months, although it was highly significant at age 18 months.

5.4 Discussion

The reason for these differences in estimates of the soil lead to dust lead pathway are not obvious. Other studies have shown that there is a strong correlation between soil lead concentration and dust lead concentration. We used both dust lead loading (mg. Pb per square meter of surface) and dust lead concentration (mg Pb per g dust) in our analyses. The dust lead concentrations in the Boston study were calculated by taking the ratio of the amount of lead in the vacuum sample to the total weight of dust, but this weight could not be accurately calculated

in very small samples and the calculated concentrations show an upward bias with decreasing weight of the dust sample. In addition to being suspected of bias, the calculated floor lead concentrations were also much worse predictors of blood lead than were the dust lead loadings.

It is known that dust cleaning can significantly reduce the loading of lead on floors without changing the concentration of lead in dust (Dolcourt et al. 1981). Additional work is needed to identify the appropriate measure of lead in house dust for use in pathway models of lead exposure. None of the studies that have attempted to directly estimate the total amount of soil and dust intake by children (Binder et al. 1986, Clausing et al. 1987, van Wijnen et al. 1990, Calabrese et al. 1989, 1990, 1991ab, and Davis et al. 1990) have explicitly adjusted these estimates for the total amount of dust in the environment. The study by van Wijnen (1990) does show that the estimated soil and dust intake varies with the environment, by comparing a campground (high intake) with a day care center (low intake). We therefore expect to find differences in the soil lead to dust lead pathway at other urban sites as well as at rural mining and smelter sites. The differences in soil-to-dust pathways are real, though not well understood. Even if improved source identification techniques allowed estimation of the separate contributions of lead-based paint and lead in soil to household dust lead, we would still expect to find large differences in lead pathways among the housing units in a community, between communities, and possibly even differences in the soil-to-dust lead pathways between the neighborhoods within a single community. The dust lead metric used in the pathway studies should always be that one which best predicts children's blood lead, even if it is more difficult to relate this dust lead metric to soil lead.

TABLE 5-1. Auto-Regressive Structural Equation Model for Blood Lead in MODEL 1: Auto-Regressive Dust Lead Model, Asymptotic Standard Errors are Shown in Parentheses

Variable	Predicted Blood Lead at AGE OF CHILD (MONTHS) 12	18	24
Blood Lead, µg/dl 6 month earlier	0.401*** (0.103)	0.409*** (0.060)	0.418*** (0.050)
Dust Lead Loading mg/sq.m at present (a)	0.0001 NS (0.0048)	0.0842*** (0.0122)	-.---
Dust Lead Loading, mg/sq.m 6-12 Months Earlier	-.--- .0070)	0.0179** (0.0070)	0.0165+ 0.0085
Soil Lead, mg/g at present (c)	0.665*** (0.189)	0.400* (0.174)	0.167 NS (0.108)
Refinishing Within Last 6 Months (No = 0 Yes = 1)	3.25*** (1.08)	3.68*** (0.174)	1.62*** (1.108)
Refinishing 6-12 Months Earlier	-.---	-1.30+ (0.71)	-1.65 NS (0.57)
Missing Soil Lead (No = 0 Yes = 1)	0.72 NS (0.91)	0.28 NS (0.81)	1.60*** (0.50)
Measurement Std. Dev. µg/dl	5.67	3.99	2.93

KEY: NS: P > 0.10
+: P between 0.05 and 0.10
*: P between 0.01 and 0.05
**: P between 0.001 and 0.01
***: P < 0.001

TABLE 5-2. Auto-Regressive Structural Equation Models for Dust Lead Loading and Soil Lead in MODEL 1: Auto-Regressive Model for Dust Lead Loading

Variable	Predicted Variable: Dust Lead Loading (mg/sq.m) 18 Mos.	Dust Lead Loading (mg/sq.m) 24 Mos.	Soil Lead (µg/g) 24 Mos.
Dust Lead Loading mg/sq.m	0.0035 NS (0.0041)	0.1086*** (0.0325)	0.970*** (0.216)
Lag (Months)	12	6	18
Refinishing Within Last 6 Months (No = 0 Yes = 1)	5.38*** (1.95)	-1.45 NS (1.50)	0.0021 NS (0.1245)
Refinishing 6-12 Months Earlier (No = 0 Yes = 1)	-4.56** (1.37)	0.06 NS (1.39)	-0.343*** (0.094)
Soil Lead mg/g at 18 Months	1.67*** (0.20)	-.---	0.917*** (0.096)
Missing Soil Lead at 18 Months	-1.92+ (1.05)	-.---	0.529*** (0.094)
Missing Soil Lead at 24 Months (No = 0 Yes = 1)	-.---	-.---	-0.816*** (0.088)
Measurement Std. Dev. mg/sq.m or mg/g	1.38	7.75	0.683

KEY:
- NS: P > 0.10
- +: P between 0.05 and 0.10
- *: P between 0.01 and 0.05
- **: P between 0.001 and 0.01
- ***: P < 0.001

TABLE 5-3. Auto-Regressive Structural Equation Model for Soil Lead, and Models for Dust Lead Loading in MODEL 2: No Auto-Regression for Dust Lead

Variable	Predicted Variable: Dust Lead Loading (mg/sq.m) 18 Mos.	Dust Lead Loading (mg/sq.m) 24 Mos.	Soil Lead (µg/g) 24 Mos.
Dust Lead Loading mg/sq.m	-.---	-.---	0.901*** (0.245)
Lag (Months)	--	--	18
Refinishing Within Last 6 Months (No = 0 Yes = 1)	6.46*** (2.21)	-2.37 NS (1.75)	0.013 NS (0.131)
Refinishing 6-12 Months Earlier (No = 0 Yes = 1)	-5.96*** (1.56)	1.93 NS (1.61)	-0.312*** (0.100)
Refinishing 12-18 Months Earlier	-.---	-2.41 NS (1.61)	0.302*** (0.109)
Soil Lead mg/g at 18 Months	1.97*** (0.28)	0.28 NS (0.71)	0.950*** (0.100)
Missing Soil Lead at 18 Months	-0.80 NS (0.96)	-0.23 NS (1.22)	0.552*** (0.096)
Missing Soil Lead at 24 Months (No = 0 Yes = 1)	-.---	0.78 ns (1.34)	-0.763*** (0.100)
Measurement Std. Dev. mg/sq.m or mg/g	1.14	7.80	0.651

KEY:
NS: P > 0.10
+: P between 0.05 and 0.10
*: P between 0.01 and 0.05
**: P between 0.001 and 0.01
***: P < 0.001

REFERENCES

Angle CR, Marcus AH, Cheng I-H, McIntire MS. 1984. Omaha childhood blood lead and environmental lead: A linear total exposure model. Environ Res 35:160-170.

Bellinger D, Leviton A, Sloman J. 1989. Antecedents and correlates of improved cognitive performance in children exposed in utero to low levels of lead. Environ Health Persp in press

Bellinger R. 1989. Effects of lead on cognitive development: Results from the Boston Study. Presented at: Lead: A Conference on Advances in Lead Research with Related Implications for Environmental Health. National Institute of Environmental Health Sciences, RTP, NC.

Bellinger D, Leviton A, Waternaux C, Needleman H, Rabinowitz M. 1987. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. New Engl J Med 316:1037-1043.

Bellinger R. 1989. Effects of lead on cognitive development: Results from the Boston Study. Presented at: Lead: A Conference on Advances in Lead Research with Related Implications for Environmental Health. National Institute of Environmental Health Sciences, RTP, NC.

Bellinger D, Leviton A, Needleman HL, Waternaux C, Rabinowitz M. Low-level lead exposure and infant development in the first year. Neurobehav Toxic and Terat, in press.

Binder S, Sokal D, Maughan D. 1986. Estimating soil ingestion: The use of tracer elements in estimating the amount of soil ingested by young children. Arch Environ Hlth 41:341-345.

Bornschein RL, Clark CS, Pan UW, Succop PA et al. 1991. The Butte-Silver Bow Environmental Health Lead Study. Department of Environmental Health, University Cincinnati Medical Center. June 1991. 23

Bornschein RL, Clark CS, Pan UW, Succop PA et al. 1990. Midvale Community Lead Study. Department of Environmental Health, University Cincinnati Medical Center. July 1990.

Bornschein RL, Clark CS, Grote J, Peace B, Roda S, Succop P. 1988. Soil lead - Blood lead relationshipin a former lead mining town. In: Environmental Geochemistry and Health, Monograph Series 4, Lead in Soil: Issues and Guidelines. (Eds) BE Davies and BG Wixson. Science Review Limited, Northwood, England. pp. 149-160.

Bornschein RL, Krafft KM, Clarc CS et al. 1986. External surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In DD Hemphill (ed) Trace Substances in Environmental Health II. University of Missouri, Columbia MO.

Bornschein RL, Succop P, Dietrich RN, Clark CS, Que Hee S, Hammond PB. 1985. The influence of social and environmental factors on dust lead, hand lead, and blood lead levels in young children. Environ Res 38:108-118.

CDC. 1986. East Helena, Montana Child Lead Study. 1986. Lewis and Clark County Health Department and Montana Department of Health and Environmental Sciences. Centers for Disease Control, Public Health Service, US Department of Public Health and Human Services, Atlanta, GA.

CDC. 1986. Kellogg Revisited - 1983. Childhood Blood Lead and Environmental Status Report. Panhandle District Health Department and Idaho Department of Health and Welfare. Center for Environmental Health, Centers for Disease Control. US Environmental Protection Agency.

Clark S, Bornschein R, Succop P, Peace B. 1988. The Cincinnati Soil-Lead Abatement Demonstration Project. In: Environmental Geochemistry and Health Monograph Series 4. **Lead in Soil: Issues and Guidelines.** (Eds) BE Davies and BG Wixson. Science Reviews Limited, Northwood, England. pp. 287-300.

Clark CS, Bornschein RL, Succop PA et al. 1985. Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. Environ Res 38:46-53.

Clausing P, Brunekreef B, van Wijnen JH. 1987. A method for estimating soil ingestion by children. Int Arch Occup Environ Hlth 59:73-82.

Colorado Department of Health (Division of Disease Control and Environmental Epidemiology), University of Colorado at Denver (Center for Environmental Sciences), and US Dept of Health and Human Service (ATSDR/PHS). Leadville Metals Exposure Study. April 1990.

Davis S, Waller P. 1990. Quantitative estimates of soil ingestion in normal children between the ages of 2 and 7 years: Population-based estimates using aluminum, silicon and titanium as soil tracer elements. Arch Environ Hlth (in press).

Dolcourt JL, Finch C, Coleman GD, et al. 1981. Hazard of lead exposure in the home from recycled automobile storage batteries. Pediatrics 68(2):225-228.

Farfel MR, Chisolm JJ. 1991. An evaluation of experimental practices for abatement of residential lead-based paint: Report on a pilot project[1]. Environ Res 55:199-212.

Farfel MR, Chisolm JJ. 1990. Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. AJPH 80(10):1240-

Fuller WA, Hidiroglou MA. 1978. Regression estimation after correcting for attenuation. J Am Statist Assoc 73:99-104.

Fuller WA, Pantula SG. 1982. A computational algorithm for the factor model. Iowa State University, Ames, Iowa.

Fuller WA. 1987. Measurement Error Models. John Wiley & Sons, Inc., Somerset, NJ.

Fuller WA. 1978. An affine linear model for the relation between two sets of frequency counts: Response to query. Biometrics 34:517-521.

Lewis and Clark County Health Department et al. 1986. East Helena, Montana: Child Lead Study. Summer 1983. July 1986.

Marcus AH, Cohen J. 1988. Modeling the Blood Lead - Soil Lead Relationship. In: Environmental Geochemistry and Health Monograph Series 4. **Lead in Soil: Issues and Guidelines.** (Eds) BE Davies and BG Wixson. Science Reviews Limited, Northwood, England. pp. 161-174.

Rabinowitz M, Bellinger DC. 1988. Soil lead-blood lead relationship among Boston children. Bull Environ Contam Toxicol 41:791-797.

Rabinowitz M, Leviton A, Bellinger D. 1985. Home refinishing, lead paint, and infant bloodlead levels. Am J Public Health 75:403-404.

Rabinowitz M, Leviton A, Needleman H, Bellinger D, Waternaux C. 1985. Environmental correlates of infant blood lead levels in Boston. Environ Res 38:96-107.

Rabinowitz MB, Wetherill GW, Kopple JD. 1976. Kinetic analysis of lead metabolism in healthy humans. J Clin Invest 58:260-270.

Rabinowitz M, Needleman H. 1983. Gasoline lead sales and umbilical cord blood lead levels in Boston, Massachusetts. Lancet 2:(8314), 63.

Rabinowitz MB, Kopple JD, Wetherill GW. 1980. Effect of food intake and fasting on gastrointestinal lead absorption in humans. Am J Clin Nutr 33:1784-1788.

Rabinowitz M, Needleman H. 1985. Lead in milk and infant blood: A dose-response model. Arch Environ Hlth 40(5):283-285.

Succop PA, O'Flaherty, Bornschein RL, et al. 1987. A kinetic model for estimating changes in the concentration of lead in the blood of young children. In: **International Conference: Heavy Metals in the Environment,** (Eds) Lindberg SE, Hutchinson TC. New Orleans, September 1987. (EP Consultants Ltd., Edinburgh, pp. 289-291).

USEPA. 1991a. Three City Urban Soil-Lead Demonstration Project - Midterm Project Update. USEPA ECAO/HEARD. 21S2001.

USEPA. 1986. Air Quality Criteria for Lead Volume I - IV. Environmental Criteria and Assessment Office, Office of Research and Development, RTP, NC. EPA 600/8-83-028 a-d.

van Wijnen JH, Clausing P, Brunekreef B. 1990. Estimated soil ingestion by children. Environ Res 51:147-162.

Waternaux C, Laird NM, Ware JH. 1986. Methods for analysis of longitudinal data blood lead concentrations and cognitive development. Technical Report, Harvard University School of Public Health, Biostatistics Department.

Yaffe Y et al. 1983. Identification of lead sources in California children using the stable isotope ratio technique. Arch Environ Hlth 38(4):237-245.

APPENDIX A

COMPARISON OF STATISTICAL METHODS FOR FITTING STRUCTURAL EQUATION MODELS FOR LEAD

STRUCTURAL EQUATION MODELS FITTED TO THREE DATA SETS

Ron Menton, David Burgoon, and Allan Marcus

1.0 INTRODUCTION

Lead contamination of environmental media such as soil, water, air, and house dust threatens us by placing lead in close proximity. Lead is toxic to all species tested: no known physiological process requires it for continued, normal functioning. Young children are particularly vulnerable due, in part, to their developing neurological systems. Elevated lead exposure, characterized by biological indicators such as the concentrations of lead in whole blood, skeleton, or teeth of children, have been associated with permanent impairment of their intelligence, motor skills and hearing. An important goal, therefore, is to understand the pathways by which environmental lead may enter the child's body from the air, soil, paint, dust, drinking water, and diet.

There are a variety of techniques available for analyzing environmental lead pathways. Analysis is complicated by the nature of the variables being measured and the myriad of possible confounding effects. Battelle (Ref. 1) has conducted extensive analysis of environmental lead pathways using linear structural equation models. These models were fitted to the data from three cross-sectional lead studies using the BMDP structural equations program EQS (Ref. 2). EQS estimates the parameters of linear structural equations using the Bentler and Weeks model (Ref. 3). One of the underlying assumptions of the linear structural equations models is that the response variables are normally distributed. Because environmental lead and blood lead levels both have highly skewed distributions this assumption may not be appropriate. A more appropriate model for estimating

blood lead levels from environmental lead levels may be the log-additive model, in which the geometric mean of blood lead is modelled as a function of the additive effects of the environmental lead levels. In this task, the nonlinear regression procedure (Proc Model) in the Statistical Analysis System (SAS) program was used to fit the log-additive model to the data from three cross-sectional studies. Results and parameter estimates calculated for linear systems of equations and log-additive systems of equations were compared for each of the data sets.

Three data sets from studies investigating environmental lead and its impact on children's blood lead concentrations were received. The data sets are from studies of populations in Kellogg, Idaho; East Helena, Montana; and Midvale, Utah. In each case, a primary lead smelter had operated in the vicinity; the East Helena smelter is still in operation. Original data files were stored as SYSTAT databases and were converted to SAS data sets using the program DBMSCOPY. To facilitate the analysis to follow, listings of the variables available in each data set were obtained. These lists are shown in Tables A-1 to A-3 in Appendix A. A description accompanies most variable names, though we were unable to discern the nature of some of the variables.

These data sets were used for comparing results based on the log-additive model with those obtained from the linear system of equations. Several models, developed through previous work (Ref. 1) were fitted to the data sets. The models are attempts to explain the concentration of lead in a child's blood stream using a number of environmental variables and measures (e.g. the concentration of lead in the surrounding soil, the amount of lead in the house dust, or the socioeconomic status of the family). Three basic model types were fitted: a single, log-additive equation; a system of linear equations; and a system of log-additive equations.

Section 2.0 compares the results for the single equation, log-additive model. The comparison is between the

parameter estimates previously obtained (Ref. 1) using the SAS procedure, PROC NLIN, and the parameter estimates produced by us using the PROC MODEL procedure. Section 3.0 outlines our results for the system of linear equations model, generated using the PROC MODEL procedure. These results are compared to those previously calculated (Ref. 1) using the AGLS procedure available in the EQS program from BMDP. Section 4.0 examines the system of log-additive equations model, again produced using the PROC MODEL procedure. The resulting parameter estimates are compared to those generated for the system of linear additive equations.

2.0 THE SINGLE EQUATION, LOG-ADDITIVE MODEL

Lead concentration measures such as blood, dust, or soil are often skewed. A log transformation of the variables can often rectify this, but a loglinear model is inappropriate for environmental lead pathway analysis. A loglinear model would produce an estimate of zero blood lead concentration if any of the independent variables were measured to have a near zero concentration, regardless of the magnitude of the other independent variables. It is highly unlikely a child's blood lead concentration would be nearly zero were the child to be exposed to highly elevated levels of lead in soil and dust, while being spared exposure to lead in air or water.

An alternative approach, which we call the log-additive model, has been used by EPA (US EPA 1986, 1989a) and in other studies (Marcus and Bernholc 1990; Angle et al. 1984). This model entails fitting the same linear additive equation, with both sides of the equation log transformed and error assumed to be multiplicative rather than additive. The log-additive model we fitted is as follows,

where,

PbB = Concentration of Lead in Blood,

PbS = Concentration of Lead in Soil,

PbD = Concentration of Lead in House Dust,
PbA = Concentration of Lead in Air,
INCOME = Measure of Family's Income,
PbDmiss = Indicator that PbD measure is missing,
PbSmiss = Indicator that PbS measure is missing, and
$\in$ = Unmeasured error.

The two missing value indicator variables are included to estimate the average blood lead increment of the missing cases (Marcus and Bernholc 1990). This model was fitted to the data sets from Kellogg and East Helena. Table 1 compares the parameter estimates calculated previously (Marcus and Bernholc 1990), using the PROC NLIN procedure, with those calculated by the PROC MODEL procedure.

3.0 SYSTEM OF LINEAR ADDITIVE EQUATIONS

The system of linear equations model begins with a single equation for explaining the concentration of lead in a child's blood as a function of specific environmental variables (similar to that employed in the single equation, log-additive model). However, three of these variables, the concentration of lead in house dust (PbD), the concentration of lead in the surrounding soil (PbS), and the product of concentration of lead on the child's hands times the normalized frequency of mouthing non-food objects (PbDXM), are further expressed as functions of other environmental variables. Specifically, the system of linear equations fitted to the East Helena, and Kellogg data sets is

where,

AGE0 = Indicates the child is less than 12 months of age,

AREA1 = Indicates the residence within first (of three) concentric rings around the neighboring smelter,

AREA2 = Indicates the residence within second concentric ring,

EATALL = Normalized measure of child's tendency to pica.

The two AREA variables are used as a surrogate for the concentration of the lead in the air. Air lead level was measured for each area represented as being contained in a concentric ring (three rings; maximum radius of 6 miles) emanating from the smelter and assumed uniform across the ring, therefore we preferred to utilize an indicator variable. A measure of the concentration of lead on the child's hands was not available, so a surrogate variable was developed. This variable (PbDXM) is the product of the concentration of lead in the house dust (PbD) and the measure of the child's tendency to put non-food objects such as toys, dirt, cigarettes, plaster etc. into his or her mouth (EATALL). These Kellogg and East Helena data sets had very similar measures available (see Appendix A), thus allowing the use of a common model. Table 2 displays the parameter estimates produced for the Kellogg and East Helena data sets using the PROC MODEL procedure. Parameter estimates previously calculated using the EQS program (Marcus and Bernholc 1990), are included for comparison.

The Midvale data set had different information available, so the model fitted to the data was slightly different. Specifically,

where,

PRE20 = Indicates the residence was built in the 19th Century,

POSTWW2 = Indicates the residence was built after World War II,

SES = Hollingshead Socioeconomic Scale (08-66) measurement for the family, and

RMVPAINT = Indicates paint has been removed from the residence in the past six months.

The variables, X and Y, are measures of the distance from the neighboring smelter. Therefore, a quadratic measure of distance from the smelter is used as a surrogate for the concentration of lead in the air. As in the Kellogg and East Helena data sets, a surrogate of hand lead is utilized: the product of a normalized tendency to pica (EATALL) and the concentration of lead in house dust (PbD). The additional information on the age of the residence (PRE20, POSTWW2), and household refinishing that may affect dust lead by removing lead-based paint but releasing lead dust during abatement (RMVPAINT) was also incorporated in the model. Table 3 compares the parameter estimates produced by the EQS program, with those calculated by the PROC MODEL procedure.

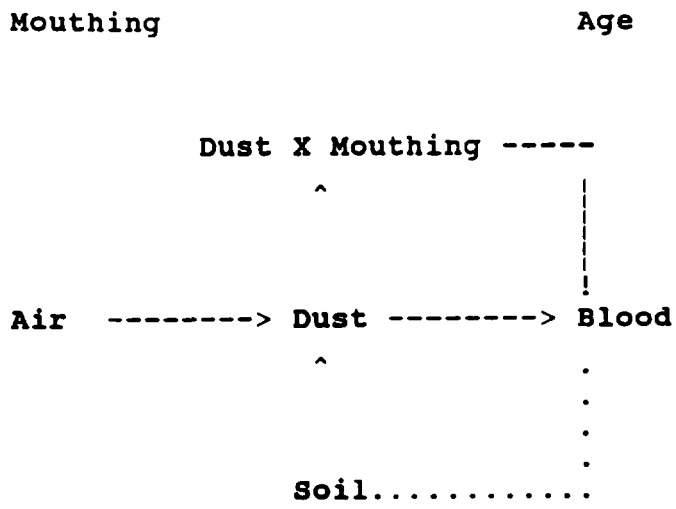
4.0 SYSTEM OF LOG-ADDITIVE EQUATIONS

This model is simply the system of equations extension of the single equation log-additive model discussed in Section 2.0. A system of log-additive equations are utilized, rather than the single dependency equation for blood lead level. Until the development of the PROC MODEL procedure, there was no relatively easy way to fit the resulting nonlinear system of equations. Log-additive models utilized the same dependence relationships the system of linear additive equations models described in Section 3.0. The error in each equation was assumed to be multiplicative, and both sides of the equation were log transformed. The system of log-additive equations fitted to the Kellogg and East Helena data sets is

Table 4 displays the parameter estimates for the log-additive model fitted to the Kellogg and East Helena data sets. These estimates are compared to those calculated for the system of linear equations using the PROC MODEL procedure, displayed in Section 3.0. Similarly, Table 5 contrasts the parameter estimates from the systems of log-additive and linear equations fit to the Midvale data set.

5.0 CONCLUSION

Different models were fitted to three data sets, using a variety of software procedures. Interestingly, the software used had minimal effect on the estimated parameters for a particular model and data set. The estimated standard errors produced by each technique were often even closer. The statistical significance of the parameter estimates did sometimes change, depending upon the software used (e.g. EQS versus PROC MODEL). Overall, the system of log-additive equations models suggested the following pathway,



The lead in soil and dust in the surrounding environment seemingly contributes to the child's blood lead concentration. The non-significance of a pathway from hand dust lead to blood lead, often cited in recent literature, may be due to the nature of the surrogate hand variable (dust * mouthing). The structure of the surrogate hand variable almost certainly is the reason for the mouthing effect on blood lead concentration. These results are not that dissimilar to those previously obtained (Marcus and Bernholc 1990) based on fitting systems of linear equations to the data using the EQS procedures.

Table A-1. East Helena, Montana data set

Variable Name	Description of the Variable	Variable Type	Number of Digits
AGE	Age of the Individual (years)	Real	4.3
AGE_SQ	Squared Age (rounded to nearest integer)	Integer	2
BLOOD	Concentration of Lead in the Blood ($\mu g/dl$)	Integer	2
AIR	Concentration of Lead in the Air ($\mu g/m^3$)	Real	3.2
DUST	Lead Concentration in Household Dust ($\mu g/g$)	Real	7.2
GARDEN	Lead Concentration in the Garden ($\mu g/g$)	Real	5.1
SOIL	Lead Concentration in the Soil ($\mu g/g$)	Real	5.1
LNBLOOD	Natural Log of Lead Concentration in Blood	Real	4.3
LNDUST	Natural Log of Pb Concen. in Household Dust	Real	4.3
LNGARDEN	Natural Log of Pb Concen. in Garden	Real	4.3
LNSOIL	Natural Log of Pb Concen. in Soil	Real	4.3
AREA	Area of Residence (in reference to smelter) (1, less than 1.6 km from smelter; 2, 1.6-4.0 km; 3, more than 4.0 km)	Category	1
AREA1	Area Indicator (1 if AREA=1, 0 else)	Indicator	1
AREA2	Area Indicator (1 if AREA=2, 0 else)	Indicator	1
FEP		Integer	2
IDNUMX	Individual Indentification Sequence	Character	7
INCOME	CDC Income Index (1, < $10000/yr; 2, ; 3,)	Category	1
STATE	Value is always 2	Constant	1
SMOKET	Is a smoker present? (1=Yes, 0=No)	Indicator	1
PbDXMOBBY	Does a household member have a hobby involving lead? (1=Yes, 0=No)	Indicator	1
CPOHR	Hours Individual Play Outdoors	Integer	2
COUTTT	Hours Individual Spends Away From Home	Integer	2
CORIFL	Hours Individual Plays on the Floor	Integer	2
TOYS	(0, 1)	Indicator	1
PERSNUM	(1, 2, 3)	Category	1
STRMWIND	(0, 1) Indicate variable for Storm Windows	Indicator	1
CORIFU	Does the Individual Mouth on Furniture? (3, A Lot; 2, Just Once in a While; 1, Almost Never)	Category	1
CORIPA	Does Individual Put Paints Chips in Mouth? (3, A Lot; 2, Just Once in a While; 1, Almost Never)	Category	1
CORIOT	Does Individual Put Other Things in Mouth? (3, A Lot; 2, Just Once in a While; 1, Almost Never)	Category	1
CORISW	Does the Individual Swallow Non-Food Items? (3, A Lot; 2, Just Once in a While; 1, Almost Never)	Category	1
COUTWT	Individual Often Takes Food Outside? (1=Yes, 0=No)	Indicator	1

Table A-1. East Helena, Montana data set
(Continued)

Variable Name	Description of the Variable	Variable Type	Number of Digits
WASH	Does the Individual Have His/Her Hands or Face Washed? (3, Rarely; 2, Occasionally; 1, Usually; 0, Almost Always)	Category	1
PLAYT2	Individual Plays... (1, Alone or With Other Children; 0, Mostly With Adults)	Indicator	1
CPGTYPE	Individual Plays on Ground that Is... (1=Not Grassy, 0=Grassy)	Indicator	1
VEGT	Individual Often Eat Neighbourhood-Grown Vegetables? (1=Yes, 0=No)	Indicator	1
VITA	Does the Individual Take Vitamins? (1=Yes, 0=No)	Indicator	1
EATSNOWT	Does the Individual Sometimes Eat Snow? (1=Yes, 0=No)	Indicator	1
CPUFDT	Individual Spends Daytime Hours... (1, At Home; 0, Away From Home)	Indicator	1
EATALL	Total Arithmetic Score (01-20)	Integer	1
DUST_ALL	EATALL*DUST, Surrogate for Hand Lead Measure	Real	7.2
SOILHOUR		Real	6.1

Table A-2. Kellogg, Idaho data set

Variable Name	Description of the Variable	Variable Type	Number of Digits
AGE	Age of the Individual (years)	Real	4.3
AGE_SQ	Squared Age (rounded to nearest integer)	Integer	2
BLOOD	Concentration of Lead in the Blood ($\mu g/dl$)	Integer	2
AIR	Concentration of Lead in the Air ($\mu g/m^3$)	Real	3.2
DUST	Lead Concentration in Household Dust ($\mu g/g$)	Integer	5
GARDEN	Lead Concentration in the Garden ($\mu g/g$)	Integer	4
SOIL	Lead Concentration in the Soil ($\mu g/g$)	Integer	5
LNBLOOD	Natural Log of Lead Concentration in Blood	Real	4.3
LNDUST	Natural Log of Pb Concen. in Household Dust	Real	4.3
LNGARDEN	Natural Log of Pb Concen. in Garden	Real	4.3
LNSOIL	Natural Log of Pb Concen. in Soil	Real	4.3
FEP	Free Erythrocyte Protoporphyrin, ug/dl	Integer	2
IDNUMX	Individual Indentification Sequence	Character	7
INCOME	CDC Income Index (1, < $10000/yr; 2, ; 3,)	Category	1
STATE	Value is always 1	Constant	1
AREA	Area of Residence (in reference to smelter) (1, less than 1.6 km from smelter; 2, 1.6-4.0 km; 3, more than 4.0 km)	Category	1
AREA1	Area Indicator (1 if AREA=1, 0 else)	Indicator	1
AREA2	Area Indicator (1 if AREA=2, 0 else)	Indicator	1
PbHOBBY	Does a Household Member Have a Hobby Involving Lead? (1=Yes, 0=No)	Indicator	1
PERSNUM	(1, 2, 3)	Category	1
SMOKET	Is there a smoker present? (1=Yes, 0=No)	Indicator	1
STRMWIND	(0, 1) Indicate variable for Storm Window	Indicator	1
TOYS	? (0, 1)	Indicator	1
COUTTT	Hours Individual Spends Away From Home?	Integer	2
CPOHR	Hours Individual Plays Outdoors?	Integer	2
CORIFL	Hours Individual Plays on the Floor?	Integer	2
CFAVOR	All Data is Missing		
CORIFU	Does the Individual Mouth on Furniture? (3, A Lot; 2, Just Once in a While; 1, Almost Never)	Category	1
CORIOT	Does Individual Put Other Things in Mouth? (3, A Lot; 2, Just Once in a While; 1, Almost Never)	Category	1
CORIPA	Does Individual Put Paint Chips in Mouth? (3, A Lot; 2, Just Once in a While; 1, Almost Never)	Category	1
CORISW	Does the Individual Swallow Non-Food Items? (3, A Lot; 2, Just Once in a While; 1, Almost Never)	Category	1

Table A-2. Kellogg, Idaho data set
(Continued)

Variable Name	Description of the Variable	Variable Type	Number of Digits
COUTWT	Individual Often Takes Food Outside? (1=Yes, 0=No)	Indicator	1
CPGTYPE	Individual Plays on Ground that is... (1=Not Grassy, 0=Grassy)	Indicator	1
CPUFDT	Individual Spends Daytime Hours... (1, At Home; 0, Away From Home)	Indicator	1
EATSNOWT	Does the Individual Eat Snow? (1=Yes, 0=No)	Indicator	1
ORAL	Does Individual Use Pacifier, Suck Thumb, or Chew Fingernails? (1=Yes, 0=No)	Indicator	1
PLAYT2	Individual Plays... (1, Alone or With Other Children; 0, Mostly With Adults)	Indicator	1
VEGT	Individual Often Eat Neighbourhood-Grown Vegetables? (1=Yes, 0=No)	Indicator	1
VITA	Does Individual Take Vitamins? (1=No, 0=Yes)	Indicator	1
WASH	Does the Individual Have His/Her Hands or Face Washed? (3, Rarely; 2, Occasionally; 1, Usually; 0, Almost Always)	Category	1
EATALL	Total Arithmetic Score (01-20)	Integer	2
DUST_ALL	EATALL*DUST, Surrogate for Hand Lead Measure	Integer	5
SOILHOUR		Integer	6
XDUST		Integer	5
DUST_MIS	Is DUST (Pb Concentration in Household Dust) Measurement Missing? (1=Yes, 0=No)	Indicator	1
EAT_MIS	(0, 1)	Indicator	1
XDUSTALL	= DUSTALL if not missing, 0 otherwise	Integer	5
LDUSTALL	Natural Log of the Variable XDUSTALL	Real	5.3
DUSTBALL	(0, 1)	Indicator	1

Table A-3. Midvale, Utah data set

Variable Name	Description of the Variable	Variable Type	Number of Digits
ID	Individual Identification Number	Integer	3
SECTION	Section of the City (25, NE; 26, NW; 35, SW; 36, SE)	Integer	2
BLOCK	Block of the City	Integer	3
APT	Apartment Number	Symbol	3
PBB	Concentration of Lead in Blood (μg/dl)	Real	2.1
LOGPBB	Natural Log of Concen. of Lead in Blood	Real	4.3
AGE	Age of the Individual (months)	Integer	2
SEX	Sex of the Individual	Indicator	1
SIBCOUNT	Number of Siblings	Integer	1
RACE	Race of the Individual (2, Caucasian; 3, Hispanic; 4, Asian; 5, Native American)	Category	1
WIC	Does Individual Participate in WIC Program? (1=Yes, 0=No)	Indicator	1
PAC	Does the Individual Use a Pacifier? (1=Yes, 0=No)	Indicator	1
SIBCLASS	? (1, ; 2, ; 3,)	Category	1
FAMILY	Family Identification Number	Integer	3
AGECLASS	(1, ; 2, ; 3, ; 4, ; 5, ; 6,) Age Class	Category	1
ETHNIC	(1, ; 2, ; 3, ; 4,) Ethnic Group	Category	1
INCLUDE	Indicate variable for Data Completeness	Indicator	1
SES	Hollingshead Scale for Socio-Economic Status (Low=8, High=66)	Integer	2
Y	North-South Coordinate with 0 at SE Corner of Section 35 (+, North; -, South)	Real	4.3
X	East-West Coordinate with 0 at SE Corner of Section 35 (+, East; -, West)	Real	4.3
DISTANCE	Distance from Smelter (=SQRT(X^2+Y^2))	Real	4.32
XX	X in km	Real	4.3
YY	Y in km	Real	4.3
X2	XX Variable Squared	Real	4.3
Y2	YY Variable Squared	Real	4.3
XY	Product of Variables, XX*YY	Real	4.3
PBSP	Pb Concen. in Soil, Perimeter (ppm)	Integer	4
PBSG	Pb Concen. in Soil, Garden (ppm)	Integer	4
PBSB	Pb Concen. in Bare Soil (ppm)	Integer	4
PBSS	Pb Concen. in Soil, Play Area in Yard (ppm)	Integer	3
PBDX	Pb Concen. in Dust at Entrance (ppm)	Integer	4
PBDI	Pb Concen. in Dust in the Interior, Composite of Three Measurements (ppm)	Real	5.1

Table A-3. Midvale, Utah data set
(Continued)

Variable Name	Description of the Variable	Variable Type	Number of Digits
PBW	Lead Concentration of the Tap Water (first 250ml)	Real	3.1
XRFX	Maximum X-Ray Fluorescence Measure of Exterior Surface (mg/cm^2)	Real	5.3
XRFI	Maximum X-Ray Fluorescence Measure of Interior Surface (mg/cm^2)	Real	5.3
LOGPBSP	Natural Log of Pb Concen., Peripheral Soil	Real	4.3
LOGPBSG	Natural Log of Pb Concen., Garden Soil	Real	4.3
LOGPBSB	Natural Log of Pb Concen., Bare Soil	Real	4.3
LOGPBSS	Natural Log of Pb Concen., Usual Play Area	Real	4.3
LOGPBDX	Natural Log of Pb Concen., Dust at Entrance	Real	4.3
LOGPBDI	Natural Log of Pb Concen., Dust in Interior	Real	4.3
LOGXRFX	Natural Log of XRF of Exterior Surface	Real	4.3
LOGXRFI	Natural Log of XRF of Interior Surface	Real	4.3
PRE20	Was the Home Built Before 1900? (1=Yes, 0=No)	Indicator	1
POSTWW2	Was the Home Built After World War II? (1=Yes, 0=No)	Indicator	1
RESTIME	Child's Duration in Residence (months)	Integer	2
FLOOR	Main Floor on which Family Resides (0, Basement; 1, Ground Level)	Integer	1
RMVPAINT	Has Paint Been Removed In Last Six Months? (1=Yes, 0=No)	Indicator	1
VEGGARDN	Does Residence Have a Vegetable Garden? (1=Yes, 0=No)	Indicator	1
FRUGARDN	Does Residence Have a Flower Garden? (1=Yes, 0=No)	Indicator	1
CROPS	Does Residence Have Root Crops? (1=Yes, 0=No)	Indicator	1
SOILFILL	Has Soil Been Brought into the Yard? (1=Yes, 0=No)	Indicator	1
PGARDEN	Does the Individual Play in the Garden, While Work Being Done (1=Yes, 0=No)	Indicator	1
AC	Does the Residence Have Air Conditioning? (1=Yes, 0=No)	Indicator	1
THUMB	Does the Individual Suck His/Her Thumb or Fingers? (1=Yes, 0=No)	Indicator	1
INYARD	Does the Individual Take Bottle, Pacifier or Food Into the Yard? (1=Yes, 0=No)	Indicator	1

Table A-3. Midvale, Utah data set
(Continued)

Variable Name	Description of the Variable	Variable Type	Number of Digits
FOLKMEDS	Is Ethnic Folk Medicine Ever Used? (1=Yes, 0=No)	Indicator	1
HOURS	Number of Hours Spent Away From Residence (e.g. at Daycare) Per Week (hours)	Integer	2
EATTOYS	Does the Individual Mouth on Toys? (0, Practically Never; 1, Once a Month; 4, Once a Week)	Category	1
EATSNOW	Does the Individual Eat Snow? (0; 1; 4)	Category	1
EATPAPER	Does the Individual Eat Paper? (0; 1; 4)	Category	1
EATDIRT	Does the Individual Eat Dirt? (0; 1; 4)	Category	1
EATCIGS	Does the Individual Eat Cigarette Butts? (0; 1; 4)	Category	1
EATPLSTR	Does the Individual Eat Plaster? (0; 1; 4)	Category	1
EATCHIPS	Does the Individual Eat Paint Chips? (0; 1; 4)	Category	1
EATOTHER	Does the Individual Eat Other Inappropriate Objects? (0; 1; 4)	Category	1
EATALL	Total of Previous Questions on Pica	Integer	2
AGE0	Age Indicator (1 if AGE=06-17, 0 else)	Category	1
AGE2	Age Indicator (1 if AGE=18-29, 0 else)	Category	1
AGE3	Age Indicator (1 if AGE=30-41, 0 else)	Category	1
AGE4	Age Indicator (1 if AGE=42-53, 0 else)	Category	1
AGE5	Age Indicator (1 if AGE=54-65, 0 else)	Category	1
AGE6	Age Indicator (1 if AGE=66-77, 0 else)	Category	1
AGE7	Age Indicator (1 if AGE=78-, 0 else)	Category	1
PBSPSYMB	Plotting Symbol for PBSP	Symbol	1
PBSGSYMB	Plotting Symbol for PBSG	Symbol	1
PBSBSYMB	Plotting Symbol for PBSB	Symbol	1
PBSSSYMB	Plotting Symbol for PBSS	Symbol	1
PBDXSYMB	Plotting Symbol for PBDX	Symbol	1
PBDISYMB	Plotting Symbol for PBDI	Symbol	1
XRFXSYMB	Plotting Symbol for XRFX	Symbol	1
XRFISYMB	Plotting Symbol for XRFI	Symbol	1
PBBSYMB	Plotting Symbol for PBB	Symbol	1
XRFIAGE2	XRFI * AGE2	Real	5.3
XRFIAGE3	XRFI * AGE3		1

Table A-3. Midvale, Utah data set
(Continued)

Variable Name	Description of the Variable	Variable Type	Number of Digits
DIRTAGE0	EATDIRT*AGE0 (EATDIRT if AGE0=1, 0 else) (0, Practically Never; 1, Once a Month; 4, Once a Week)	Category	1
DIRTAGE2	EATDIRT*AGE2 (EATDIRT if AGE2=1, 0 else)	Category	1
DIRTAGE3	EATDIRT*AGE3 (EATDIRT if AGE3=1, 0 else)	Category	1
DIRTAGE4	EATDIRT*AGE4 (EATDIRT if AGE4=1, 0 else)	Category	1
DIRTAGE5	EATDIRT*AGE5 (EATDIRT if AGE5=1, 0 else)	Category	1
PBDIAGE0	PBDI * AGE0	Real	5.1
PBDIAGE2	PBDI * AGE2	Real	5.1
PBDIAGE3	PBDI * AGE3	Real	5.1
PBDIAGE4	PBDI * AGE4	Real	5.1
PBDIAGE5	PBDI * AGE5	Real	5.1
PBSPAGE0	PBSP * AGE0	Integer	4
PBSPAGE2	PBSP * AGE2	Integer	4
PBSPAGE3	PBSP * AGE3	Integer	4
PBSPAGE4	PBSP * AGE4	Integer	4
PBSPAGE5	PBSP * AGE5	Integer	4
PBDIALL	EATALL*PBDI/mean(EATALL), Surrogate Hand Pb Measure	Real	7.3
LPBDIALL	Natural Log of Variable, PBDIALL	Real	4.3
XEATALL	= EATALL if not missing, 0 otherwise	Real	4.3
XPBDI	= PBDI if not missing, 0 otherwise	Real	7.3
XPBSP	= PBSP if not missing, 0 otherwise	Integer	4
MISSPBDI	Is Interior Dust Measure (PBDI) Missing? (1=Yes, 0=No)	Indicator	1
MISSPBSP	Is Perimeter Soil Measure (PBSP) Missing? (1=Yes, 0=No)	Indicator	1
XXRFI	= XRFI if not missing, 0 otherwise	Real	4.3

APPENDIX B

STATISTICAL ANALYSES OF THE BUTTE LEAD STUDY

DECEMBER, 1991

DRAFT REPORT

FOR

TASK 2-19

RELATIONSHIP BETWEEN BLOOD LEAD AND ENVIRONMENTAL LEAD FOR CHILDREN IN BUTTE, MONTANA

Allan Marcus
Task Leader

BATTELLE
Columbus Operations Arlington Office
2101 Wilson Boulevard Suite 800
Arlington VA 22201-3008

Contract 68-D0-0126

Phillip Robinson, Co-Task Manager
Karen Hogan, Co-Task Manager
Edith B. Sterrett, Project Officer

Design and Development Branch
Exposure Evaluation Division
Office of Toxic Substances
U.S. Environmental Protection Agency
Washington, DC 20460

ABSTRACT

Data from the environmental lead and child blood lead study carried out by investigators from the University of Cincinnati at Butte/Silver Bow, Montana in September 1990 (Bornschein et al. 1991) were reanalyzed by several different methods. Non-linear regression analyses found a strong and statistically significant relationship between blood lead and dust lead (adjusted for child mouthing behavior) of about 2 μg/dl in blood per 1000 μg/g lead in dust, consistent with almost all earlier studies in smelter, mining, and urban sites. The direct contribution of soil lead to blood lead was negligible. However, structural equation models showed that there was an indirect contribution of soil lead to blood lead, through a moderately strong and statistically significant relationship of soil lead near the house to interior dust lead. Exterior lead-based paint, street dust, and house age increased lead concentrations in near-surface soil cores near the house. Use of the USEPA Uptake/Biokinetic Model for Lead showed that the negligible direct role of ingested soil can be accounted for by a reduced bioavailability of soil lead, by reduced intake of soil but not household dust, or by interruption of the usual soil-to-dust pathways at Butte. Site-specific assessment of these hypothetical explanations is needed to account for the differences between Butte children and those in other lead mining and smelting towns.

RELATIONSHIPS BETWEEN BLOOD LEAD AND ENVIRONMENTAL LEAD FOR CHILDREN IN BUTTE, MONTANA

Allan H, Marcus
Battelle Memorial Institute
Research Triangle Park, NC

1.0 INTRODUCTION

Butte, Montana, has been the center of non-ferrous metal mining and smelting processes for over a century. The sources of potential environmental contamination include gold, silver, and copper mines, silver mills, smelters, and waste piles from these mines, mills, and smelters. Both historical sources of trace metal contamination and some modern sources are of concern. In 1990 investigators from the University of Cincinnati carried out a field study on trace metal contamination in the household and community environments of the Butte-Silver Bow area, and collected biological specimens from children and adults who resided there (Bornschein et al. 1991), In this paper we will describe a preliminary analysis of suspected risk factors for childhood lead exposure, where the child's venous blood lead concentration is used as the index of internal exposure.

Three methods will be used to assess the relationship between blood lead and environmental lead:

(1) Non-linear regression analysis between blood lead and a linear combination of exposure from multiple sources and pathways, where both internal and external exposure indices are log-transformed;

(2) A structural equations analysis of the relationship between blood lead and environmental lead, reaching back into the pathways from several lead sources such as paint and soil through the proximate exposure in household dust;

(3) An assessment of the plausibility of the estimated statistical relationships using EPA's Lead Model.

The first method is used to compare the blood lead relationship for the Butte data set with those derived in earlier EPA publications and reports (USEPA 1986, 1989a; Marcus and Cohen 1988; Marcus 1990, 1991). Reasons for site-specific differences in exposure or bioavailability of lead from different sources and/or pathways have been hypothesized by several investigators (Steele et al. 1990) and these differences can be tested statistically, provided that all of the data sets are analyzed using models that are internally consistent and scientifically valid. The structural equations method was presented initially by investigators at the University of Cincinnati (Bornschein et al. 1985, 1988) and has been applied by them to data from the Cincinnati Lead Program Project and from studies in Telluride CO (Bornschein et al. 1988), Midvale UT (Bornschein et al. 1990) as well as Butte (Bornschein et al. 1991).

Recent technical advances in statistical technique and computer software have allowed convenient extension of the structural equation method to models that are consistent with the other EPA analyses (Marcus 1989, 1990, 1991). A more important development is that the models presented to EPA are consistent with a detailed physical and biological representation of childhood lead exposure and biokinetics as represented by EPA's Integrated Exposure, Uptake, and Biokinetic Model for Lead (USEPA 1989b, 1990, 1991). The consistency of statistical and mechanistic models is particularly important if the relationships between blood lead and environmental lead are the basis for environmental mitigation and abatement actions. Consistency between mechanistic and statistical

models is necessary in order to be able to assert that childhood blood leads can indeed be reduced by controlling lead exposure through the appropriate pathway, or by abating lead from the appropriate source. The statistical analyses are used to identify the most likely sources and pathways for current exposure.

The use of the EPA Lead Model is two-fold. First of all, the Lead Model uses information from a very large number of studies, including other epidemiology studies, toxicology studies, and environmental media studies. The consistency of the apparent statistical results from the Butte data with prior scientific knowledge is a check on their external validity. The second reason for using the Lead Model is that it is designed to estimate changes in child blood lead over time, during intervals of changes in lead exposure following an abatement. Prediction of abatement effectiveness from cross-sectional statistical data is limited by the fact that child lead exposure changes with age over time, so that future effectiveness cannot necessarily be estimated from a current cross-sectional snapshot based on previous lead exposures.

2.0 MATERIALS AND METHODS

2.1 The Butte Data Set

Data were obtained from R. Bornschein and U.Q. Pan with information on 206 children in the 1990 Butte-Silver Bow field study. Variables in the SAS data set and constructed from that data for our analyses our shown in Table 2-1. The environmental lead variables are similar to those used in our analyses of the Midvale data (Marcus 1990, 1991). We imported these data into ASCII and SYSTAT data sets for use with other software packages.

The most important environmental lead variables characterize lead concentrations in soil and dust. PBSP denotes the soil lead concentration at the perimeter of the house, about 0.5 m from the wall. PBDCURB denotes the dust lead concentration at the curb of the street nearest to the house, PBDX denotes the concentration in dust outside the entrance of the house, and PBDI denotes the dust concentration in a composite sample from the floors. Paint lead loadings on interior and exterior surfaces are denoted by PBPIXM and PBPEXM respectively, and are the maximum X-ray fluorescence measurements on these surfaces. An water lead concentration is denoted PBW. Blood lead is denoted PbB.

Other information available includes frequency of childhood mouthing behavior observed by the caretaker, denoted MOUTH, house age denoted HOUSEAGE, scores for lead-related jobs and hobbies, family socio-economic status denoted SES, and recruitment neighborhood. The child's age and number of siblings in the family are also given. We did not randomly select a subset of children from multi-sibling families, but did eliminate 4 children whose blood lead or environmental lead data were so incomplete as to be inadequate for the statistical analyses. All other analyses were done with the 202 (in some cases, 201) remaining children.

2.2 Statistical Methods

The statistical model for the blood lead data is based on the assumption that the contribution of lead from different media and pathways is essentially additive, and nearly linear at the low

to moderately high concentrations of environmental lead to which these children were exposed. Basic biological reasons for near-additivity are given in (USEPA 1986). Non-linearity at higher concentrations may be attributable to partially-saturable absorption of lead through the gut wall (Aungst and Fung 1981), and partially-saturable binding of lead to the red blood cells (Marcus 1985c), as well as differences in chemical speciation and size of soil lead particles leading to differential lead uptake (Chaney et al. 1988). The equation fitted by non-linear least squares preserves additivity and linearity of lead sources, but is log-transformed so as to provide an approximately normal distribution of regression residual deviations:

$$\ln(PbB) = \ln(b0 + b1*PBDI + b2*PBDI*MOUTH + b3*PBSP + etc.)$$

A large number of model specifications were tested, using a backward elimination procedure for variable selection.

The structural equations models were based on prior knowledge about environmental lead pathways estimated for other lead smelter and mining communities in the Western U.S. (Marcus 1990, 1991). The coefficients were estimated using a method that is nearly free of distributional assumptions when the sample size is large enough, yet allows retention of the additive nature of lead concentrations from different sources. This method is implemented in the EQS program (Bentler 1989) and has been shown to given nearly identical results to the non-linear system model specifications fitted by SAS PROC MODEL (Marcus et al. 1991).

The simulation of the process using coefficients suggested by the statistical analyses was developed using a batch-mode version of the USEPA Lead Model version 0.6. The basis for the Lead Model is described in the Technical Source Document (USEPA 1990), the OAQPS exposure methodology staff paper (USEPA 1989a), and the Guidance Manual for the Lead Model (USEPA 1991).

3.0 RESULTS

3.1 Statistical Models for Blood Lead

The regression model for blood lead was developed in a manner analogous to those in the Lead Criteria Document (USEPA 1986). The results are shown in Table 3-1, and repeated here after the log transformation has been reversed. The estimated standard error of the coefficients is also shown in parentheses:

Blood lead (μg/dl) = 3.43 (+/- 0.42) μg/dl

+ 1.55 (+/- 0.45) PBDI mg/g ***

+ 0.36 (+/- 0.42) PBDI * MOUTH mg/g

+ 0.130 (+/- 0.184) PBSP * MOUTH mg/g

+ 0.130 (+/- 0.048) PBPIXM mg/sq.cm***

- 0.0249 (+/- 0.0100) SES **

-1.44 (+/- 0.25) [AGE < 1 YEAR] ***.

Coefficients denoted *** have significance level < 0.001, and ** denotes significance level between 0.001 and 0.01. A somewhat different model was developed using the coupled system of equations among lead variables in the structural equations model. The blood lead regression model in the structural equations model is:

Blood lead (μg/dl) = 3.03 (+/- 0.78) μg/dl
+ 1.11 (+/- 0.48) PBDI mg/g ***
+ 0.89 (+/- 0.28) PBDI * MOUTH mg/g ***
+ 0. (+/- 0.0125) PBSP mg/g
+ 0.039 (+/- 0.025) PBPIXM mg/sq.cm
- 0.0249 (+/- 0.0100) SES **

Dust lead PBDI (modified by frequency of mouthing behavior) is a significant predictor of blood lead by either method. The sum of the PBDI and PBDI*MOUTH coefficients is the total household dust effect on blood lead, which is highly similar by either method, and clearly consistent with the rate of increase of 2 μg/dl per 1000 μg/g in household dust found in most other studies (USEPA 1986; Marcus 1991). The direct effect of soil lead exposure is trivial in either model. Blood lead in children less than one year old is significantly lower than in older children. No other main effects of age were detected, although some marginally significant age interactions with dust and mouthing behavior were found. As noted in most earlier studies, blood lead is higher in families with more disadvantaged socioeconomic conditions, even when environmental lead exposure is the same.

3.2 Statistical Models for Environmental Lead Pathways

The indirect effect of soil lead and street dust on blood lead is very strong, however, since soil lead and street dust are significant sources of lead in household dust. This is shown clearly in Table 3-2, using the structural model in Figure 3-1. The regression coefficients for soil and street dust are similar by both methods. however, the larger coefficients in the structural equation model are based on connected pathways from these sources to child blood lead, thus are more relevant to child exposure.

The structural models for soil lead and exterior entrance dust lead are shown together in Tables 3-3 and 3-4. Exterior lead-based paint and house age are significant predictors of both variables. Lead in street dust may also be predictive.

3.3 Comparison With EPA Exposure/Uptake/Biokinetic Model for Lead

Effective management and abatement of environmental hazards depends upon the ability to estimate health risk from exposure data. EPA has developed an integrated model of lead exposure and biokinetics in order to estimate blood lead as a

biological indicator of lead exposure and thus estimate health risk from environmental lead (Cohen et al. 1990; USEPA 1989a, 1990, 1991). This model will be called the Lead Model. The model has been validated using epidemiologic studies at two different sites: the 1983 CDC study in East Helena, Montana, site of an active primary lead smelter; and the 1989 Univ. of Cincinnati study at Midvale, Utah, site of long-inactive smelter and mine waste piles. The statistical analyses in Sections 3.1 and 3.2 suggest that a very different set of input parameters may be needed to characterize the lead pathways from source to child in the 1990 Butte study.

Several sets of input parameters were found to provide a good prediction of the Butte child blood lead levels from environmental data for each child's current residence. The input parameters are given in Table 3-5. The criteria that were predicted are shown in Tables 3-6 through 3-8. In Table 3-5, the soil lead absorption coefficients were assumed to be very small. The "passive" absorption component P is the absorption at high concentrations that is not saturated. The "active" absorption component A is added to the "passive" component P when environmental lead concentration , denoted PbE (PBSP for soil lead, PBDI for dust lead etc.), is low. The concentration C is the value of PbE at which the active component is half as large as it is when PbE = 0.

The models in Table 3-5 represent the following situations. We assumed that the intake of household dust by children is the same at Butte as at other sites, averaging about 55 mg/day for children of ages 1 to 3 years. The total uptake of lead directly from soil appears to be low. This may be attributable either to reduced intake of lead directly from soil as discussed in Section 4, or by reduced absorption of ingested lead (i.e. bioavailability is reduced). These data cannot, by themselves, discriminate between these two possibilities. This could be specified in the Lead Model by setting the absorption fractions to 0 (P = 0 and A = 0), or by setting the combined soil and dust

intake to the dust intake alone and setting the soil/dust split to 0 soil and 100 percent dust.

Model 1 represents an effort to include at least some soil lead absorption in the Lead Model. The linear absorption model predicted high blood leads with any positive soil value, so we used the non-linear absorption model option. With no intake assumed for lead paint, fairly high absorption of lead from household dust (25 percent at low concentrations, 15 percent absorption at 1000) the soil lead absorption that adequately fitted the geometric mean was only 4 percent at low concentrations and 2 percent at 1000. Models 2-6 assumed no soil lead uptake (whether from zero absorption or zero intake). In Model 2, we assumed household dust had 30 percent absorption at all concentrations. This overpredicted the blood leads consistently, and showed a severe non-linearity in dust lead above 1000 μg/g. The non-linear dust lead absorption models 3-6 gave better results. These models allowed some intake directly from paint, 0.4 mg/day in Model 3 and 1 mg/day in Models 4-6. Since information was available on lead paint loadings by XRF, both inside and outside the house, the split between interior and exterior lead paint exposure was assumed to be 20 percent interior in Model 4, 50 percent in Model 5, and 80 percent in Model 6. To compensate for the additional uptake of lead directly from lead-based paint, in the model, a somewhat lower uptake of lead from household dust (15 percent absorption at lower concentrations) was assumed. The best-fitting model overall was Model 5: No uptake of lead from soil, 15 percent absorption of lead from house dust, and 0.5 mg/day intake of paint from both interior and exterior.

In order to achieve an adequate fit of the data, dust lead and water lead absorption estimates were reduced by trial and error.

The geometric mean blood lead (GM) based on 205 children with adequate environmental data to predict blood lead are compared with the observed blood leads in Table 3-6. Input parameters have been somewhat adjusted to match either the observed geometric mean

blood lead or the observed mean blood lead. A paired-sample t-test of the log-differences of blood lead for each child,

ln(observed blood lead/predicted blood lead),

was used as the basis for the test. All models with the specified parameters showed no mean difference between predicted and observed geometric mean blood lead. Use of the East Helena or Midvale parameters greatly over-estimated blood lead levels. It was necessary to greatly reduce soil lead absorption, and to reduce dust, water, and paint lead absorption relative to other sites, in order to estimate community mean blood lead.

The cumulative distribution functions of observed and predicted blood lead are compared in Table 3-7. All models, except Model 2, adequately describe the distribution from the 15th to the 90th percentiles. However, the lower tail is consistently over-estimated by the model, and the upper tail considerably underestimated. This is also evident from the fact that the GSD of the predicted blood leads is smaller than the observed GSD = 1.806.

The correlation between observed and predicted blood lead is shown in Table 3-8. The largest correlation between log blood leads is 0.38, and between untransformed blood leads is 0.41, both achieved in Model 5. The Lead Model accounts for about 12 percent of the variation in individual blood lead, which is consistent with the East Helena and Midvale simulations. Even a totally optimized regression model can only account for 25 to 30 percent of the variation in individual blood lead at these low levels. We have demonstrated here that the uptake of lead from soil in the Lead Model must be assumed negligible, and the uptake of lead in house dust relatively lower, than at other sites. Applications to risk management issues, such as the assessment of soil lead cleanup levels, will be described elsewhere.

4.0 DISCUSSION

The relationship between blood lead and dust lead is very similar to those we have found at other sites, about 2 μg/dl per 1000 μg/g lead in dust on the floor. However, the role of soil lead exposure in these Butte children appears to be indirect. Soil lead is a significant source of dust lead. The contribution of soil lead to house dust lead is estimated as 26.6 percent of the soil lead concentration. The contribution of soil lead at the house perimeter to dust lead at the house entrance is estimated as 70.5 percent of the lead concentration in soil. The lead in street dust at the curb also makes a significant contribution to house dust, estimated as 22 percent of the concentration of lead in dust at the curb in addition to the dripline soil lead contribution. Exterior lead-based paint makes a significant addition to soil lead contamination, therefore also indirectly contributes to lead in household dust. Thus, we may infer that exterior sources of lead make sizeable contributions to lead in household dust, and indirectly to blood lead.

The uptake of lead from Butte soils is significantly lower than at other sites, as judged by the insignificance of the soil lead regression coefficient. The uptake of lead in house dust at Butte appears to be much higher than that of soil lead, possibly due to the small size of leaded particulates in house dust compared to soil (Barltrop and Meek 1979), though somewhat lower than dust from other sites.

The use of the non-linear absorption model is inconvenient, but clearly necessary in order to improve the closeness of the fitted model as a function of soil, dust, and water lead concentration. This implies that a better regression model could have been chosen, possibly of the form

$$\ln(\text{blood lead}) = \ln(b0 + b11*PBDI + b12*PBDI/(1 + PBDI/c1) + \text{similar terms}).$$

Preliminary attempts to develop such a model ran into severe problems of non-estimable parameters. Our understanding of factors affecting the bioavailability of lead in soil and dust is too incomplete to derive a functional relationship. If the lead concentrations in soil and dust are systematically associated with differences in particle size or chemical speciation that affect bioavailability, then some non-linear relationship between blood lead and soil lead may be apparent (Chaney et al. 1988).

We cannot identify factors in the reduced uptake of lead from soil in Butte children without supporting data. The most useful of these data would be lead loadings on the child's hands. If hand lead loadings after outside play were low, then the reduced uptake of soil lead would reflect reduced intake, as might be expected if soil lead particles were too large to readily adhere to the hands. If hand lead loadings were high, then it would be easier to conclude that the bioavailability of lead in soil were low. The estimates of bioavailability of powdered lead soils in animal feeding studies may not completely reflect the realities of child exposure to present-day leaded soils. Such studies will certainly not reflect the potential hazard in converting leaded soils into fine particles that are readily transported into the household dust. Normal human activities -- gardening, excavation, play by children or pets -- will have the potential of converting even highly consolidated soil particles into finer particles. If lead is carried into household dust at sufficiently high concentrations, then the dust will pose a significant source of exposure. The quantitative similarity between blood lead uptake from household dust in Butte and at other sites suggest that differences in chemical species and sources of lead in household dust at different sites become relatively unimportant when particle size is sufficiently small.

Some of the children in Butte had blood lead concentrations that are now considered elevated -- 22.5 and 25 μg/dl. No simulation model that adequately describes typical exposures associated with a geometric mean blood lead of 4 μg/dl

can be expected to predict such atypical cases. These cases may be attributable to unusual sources of exposure, unusually large intakes of some exposure medium such as soil, paint, or water, or distinct biokinetic parameters in addition to absorption. The presence of elevated levels of lead in soil, dust, paint, and water in some of the Butte residences should be considered as potential risk factors for future residents in these housing units, as well as for children currently residing there.

TABLE 3-1. Regression Models for Blood Lead vs. Environmental Lead

Variable	Nonlinear Least Squares	Structural Equation
PBDI (mg/g)	1.547 (± 0.448)***	1.105 (± 0.475)***
PBDI * MOUTH (mg/g)	0.370 (± 0.410)NS	0.886 (± 0.282)***
PBSP (mg/g)	0.084 (± 0.120)NS	0. (± 0.0125)CON
PBP/XM (mg/sq.cm)	0.1305 (± 0.0479)***	0.0390 (± 0.0252)NS
AGE <1 Year	-1.44 (± 0.25)***	-2.06 (± 0.29)***
SES	-0.0249 (± 0.0100)**	-0.0316 (± 0.0091)***
PBDI + PBDI * MOUTH (mg/g)	**1.917 (± 0.430)*** **	**1.991 (± 0.414)*** **

KEY: NS: not statistically significant
**: P between 0.01 and 0.001
***: P < 0.00
CON: constrained estimate

TABLE 3-2. Regression Models for Dust Lead (μg/g)

Variable	Nonlinear Least Squares	Structural Equation
PBSP (μg/g)	0.242 (± 0.059)***	0.266 (± 0.040)***
PBP/XM (mg/sq.cm)	0.790 (± 1.72)NS	0.350 (± 3.76)NS
HOUSE AGE (Years)	18.03 (± 8.15)*	16.15 (± 7.05)*
PBDCURB (μg/g)	0.185 (± 0.120)NS	0.220 (± 0.115)+

KEY:
- NS: P > 0.10
- +: P between 0.05 and 0.10
- *: P between 0.01 and 0.05
- **: P between 0.001 and 0.01
- ***: P < 0.001

TABLE 3-5. Input Parameters for Soil, Dust, Paint, and Water Lead Absorption in Six Good-Fitting Models for Butte Lead Exposure

	SOIL			DUST			PAINT ABSORPTION			PAINT INTAKE	
Model	P	A	C	P	A	C	P	A	C	Total	Indoor Fract.
1	.00	.04	1000	.05	.20	1000				.00	
2	.00	.00		.30	.00					.00	
3	.00	.00		.05	.20	1000	.01	.05	1000	0.4	0.5
4	.00	.00		.03	.12	1000	.01	.01	1000	1.0	0.2
5	.00	.00		.03	.12	1000	.01	.01	1000	1.0	0.5
6	.00	.00		.03	.12	1000	.01	.01	1000	1.0	0.8

KEY: P = Passive diffusion absorption coefficient
A = Active absorption (facilitated diffusion) coefficient
C = Concentration at which active absorption is 50% saturated, in μg/L in gut.
* = Equivalent to no soil lead intake.

TABLE 3-3. Regression Models for Soil Lead (μg/g)

Variable	Nonlinear Least Squares	Structural Equation
PBPEXM (mg/sq.cm)	18.80 (± 9.27)*	29.34 (± 5.08)***
HOUSE AGE (Decades)	49.0 (± 16.3)***	70.0 (± 11.5)***
PBDCURB (μg/g)	0.679 (± 0.321)*	0.495 (± 0.226)*

KEY:
- NS: P > 0.10
- +: P between 0.05 and 0.10
- *: P between 0.01 and 0.05
- **: P between 0.001 and 0.01
- ***: P < 0.001

TABLE 3-4. Regression Models for Exterior Dust Lead at House Entrance (μg/g)

Variable	Nonlinear Least Squares	Structural Equation
PBSP (μg/g)	0.594 (± 0.113)***	0.705 (± 0.155)***
PBPEXM (mg/sq.cm)	18.8 (±16.1)NS	34.4 (± 15.5)*
HOUSE AGE (Decades)	1.35 (± 10.1)NS	-19.4 (± 21.3)NS
PBDCURB (μg/g)	0.513 (± 0.300)+	0.227 (± 0.350)NS

KEY:
- NS: P > 0.10
- +: P between 0.05 and 0.10
- *: P between 0.01 and 0.05
- **: P between 0.001 and 0.01
- ***: P < 0.001

TABLE 3-6. Comparison of Geometric Mean Blood Lead Level for Butte Predicted by Lead Model Using Six Sets of Input Parameters

Model	Geom Mean (μg/dl)	Mean (μg/dl)	GSD	P*
1	3.715	4.071	1.593	0.324
2	4.910	5.525	1.582	0.000
3	3.724	4.074	1.517	0.286
4	3.833	4.376	1.652	0.090
5	3.765	4.229	1.611	0.204
6	3.657	4.087	1.593	0.535
Observed	3.563	4.276	1.806	0.000

* Significance sample for paired t-test of equality of logarithm of geometric mean of observed and predicted blood lead.

TABLE 3-7. Comparison of Cumulative Distribution Function and Percentiles of Blood Lead Levels Predicted by Lead Model Using Six Sets of Input Parameters

Model	Percentiles					Kolmogorov-Smirnov	
	25	50	75	90	Max	Distance	P*
1	2.57	3.50	5.00	6.83	11.40	0.166	0.007
2	3.55	4.64	6.27	8.97	30.98	0.308	0.000
3	2.61	3.68	4.89	6.69	11.44	0.171	0.005
4	2.54	3.58	5.64	7.40	15.83	0.122	0.094
5	2.54	3.64	5.38	7.26	11.71	0.122	0.094
6	2.49	3.52	5.40	6.65	11.44	0.122	0.094
Observed	2.5	3.5	5.0	7.0	25.0	---	---

TABLE 3-8. Correlation of Observed and Predicted Blood Lead Levels Using Six Sets of Input Parameters in the Lead Model

Model	Correlation	
	Log-Transformed	Not Transformed
1	0.33	0.36
2	0.31	0.37
3	0.36	0.40
4	0.37	0.40
5	0.38	0.41
6	0.37	0.38

REFERENCES

Aungst BJ, Fung H. 1981. Kinetic characterization of in vitro lead transport across the rat small intestine. Toxicol Appl Pharmacol 61:39-57.

Barltrop D, Meek F. 1979. Effect of particle size on lead absorption from the gut. Arch Environ Health 34:280-285.

Bornschein RL, Clark CS, Pan UW, Succop PA et al. 1991. The Butte-Silver Bow Environmental Health Lead Study. Department of Environmental Health, University Cincinnati Medical Center. June 1991.

Bornschein RL, Clark CS, Pan UW, Succop PA et al. 1990. Midvale Community Lead Study. Department of Environmental Health, University Cincinnati Medical Center. July 1990.

Bornschein RL, Clark CS, Grote J, Peace B, Roda S, Succop P. 1988. Soil lead - Blood lead relationshipin a former lead mining town. In: Environmental Geochemistry and Health, Monograph Series 4, Lead in Soil: Issues and Guidelines. (Eds) BE Davies and BG Wixson. Science Review Limited, Northwood, England. pp. 149-160.

Bornschein RL, Krafft KM, Clarc CS et al. 1986. External surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In DD Hemphill (ed) Trace Substances in Environmental Health II. University of Missouri, Columbia MO.

Bornschein RL, Succop P, Dietrich RN, Clark CS, Que Hee S, Hammond PB. 1985. The influence of social and environmental factors on dust lead, hand lead, and blood lead levels in young children. Environ Res 38:108-118.

Chaney RL, Mielke HW, Sterrett SB. 1988. Speciation, Mobility and Bioavailability of Soil Lead. In: Environmental Geochemistry and Health, Monograph Series 4, **Lead in Soil: Issues and Guidelines.** (Eds) BE Davies and BG Wixson. Science Review Limited, Northwood, England. pp. 105-130.

Cohen J, Marcus A, Elias R. 1990. Estimating childhood multi-media lead exposure: Expanded exposure/uptake/biokinetic model. Presented at the 83rd Annual Meeting & Exhibition, Air & Water Management Assoc. Pittsburgh, PA. June 24-29, 1990.

Marcus AH. 1991. Relationship between soil lead, dust lead, and blood lead over time: A reanalysis of the Boston lead data. (manuscript in preparation)

Marcus AH, Bernholc A. 1990. Variability of household water lead levels in American cities. Report from Battelle Arlington Operations to USEPA. Contract No. 68-D8-0115.

Marcus AH, Cohen J. 1988. Modeling the Blood Lead - Soil Lead Relationship. In: Environmental Geochemistry and Health Monograph Series 4. **Lead in Soil: Issues and Guidelines.** (Eds) BE Davies and BG Wixson. Science Reviews Limited, Northwood, England. pp. 161-174.

Marcus AH. 1985. Multicompartment kinetic model for lead: III. Lead in blood plasma and erythrocytes. Environ Res 36:473-489.

Steele MJ, Beck BD, Murphy BL. 1990. Assessing the contribution from lead in mining wastes to blood lead. Reg Toxicol Pharm, 11:158-190.

USEPA. 1991. Three City Urban Soil-Lead Demonstration Project - Midterm Project Update. USEPA Environmental Criteria and Assessment Office. USEPA 21S-2001.

USEPA. 1990. Technical Support Document on Lead. USEPA Environmental Criteria and Assessment Office, Cincinnati, OH. Report No. ECAO-CIN-757.

USEPA. 1989. Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation. USEPA Office of Air Quality Planning and Standards, RTP, NC. EPA-450/2-89/011.

USEPA. 1989. Guidance Concerning Soil Lead Cleanup Levels at Superfund Sites. Directive #9355.4-02. Office of Solid Waste and Emergency Response. September 1989.

USEPA. 1986. Air Quality Criteria for Lead Volume I - IV. Environmental Criteria and Assessment Office, Office of Research and Development, RTP, NC. EPA 600/8-83-028 a-d.